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Editorial

With this issue, we bring to an end Volume XVIII. It has had, we feel, both good and bad points, though we sincerely hope that the latter have been obscured by the former in the eyes of our readers. One very bad feature, common to the JOURNAL this year as well as former years, has been the paucity of articles of interest to the pre-clinical years. When we consider that a good half of our potential readers cannot make head nor tail of the material, we believe we are guilty of straight malpractice. How this can be remedied is a knotty problem indeed, but one certainly worthy of solution.

Our last plea is that the graduating class this year and every year subscribe to the JOURNAL. If you admire your medical school and faculty at all, and most of us do, it is certainly one way of keeping in touch with "Old Toronto." Mr. N. B. G. Taylor, who did such a fine job as Associate Editor this year, will be next year's Editor-in-Chief, and with him at the helm, the JOURNAL should be a good one. Bouquets are also due to each and every one of the staff, and to Mr. Weir, our printer of the University Press.

And now, success to everybody on the exams—may Allah be kind to each and every one. We cannot help but close with a quotation from Stevenson:

"To travel hopefully is a better thing than to arrive,
And the true success is to labor."

In Memoriam

SIR FREDERICK BANTING,

K.B.E., M.C., LL.D. Qu., West., D.Sc. Tor., Yale, N.Y., F.R.S., F.R.C.P. Lond., F.R.C.S. Eng., F.R.S.C., F.R.C.S.(C).

Professor of Medical Research and Major in the Canadian Army Medical Corps.

Major Sir Frederick Banting died, in his fiftieth year, while engaged in carrying information on the results of medical research on war problems from Canada to England.

Frederick Grant Banting began his career in medical research with a mental equipment which enabled him to surmount all obstacles, to become Canada's foremost scientist and one of the world's great benefactors. His scientific curiosity was insatiable and his energy in the pursuit of knowledge boundless. His thorough training as a practical surgeon served him well, but his interest in the fundamental aspects of all medical problems was never dimmed.

Banting was a good student, but he stood apart from his fellows by virtue of his critical interest in the physical and chemical bases for the current medical teaching rather than for his ability to secure high marks in examinations. He was interested in the practical aspects of medical work and preserved throughout his career a compelling desire to relieve the suffering of individual patients. His experiences as a battalion medical officer made a lasting impression on his mind. Again and again, even in the busiest phases of the insulin investigations, he found time to set a fracture or perform a surgical operation on one of his army comrades or on some patient who was in need. His predominant thought, even in his last hour, would be to do his utmost to relieve the suffering of others.

In the insulin investigations, which made his reputation, he demonstrated many of the outstanding traits of his character. He was determined that his idea should have an adequate trial. The difficulties were surmounted with a dogged perseverance which would have achieved results in any line of endeavour. His determination was a predominant factor in the success which was secured. His comradeship when he was an unknown young investigator and his generosity in sharing the credit of the discovery of insulin have placed the writer forever in his debt.

After the discovery of insulin he interested himself in diabetic patients for a time but soon returned to his scientific investigations. With other colleagues he made determined assaults on the walls of ignorance which obscure our view. He contributed to our knowledge of the suprarenal cortex. He established a reputation for himself in cancer research. His

laboratory facilitated the studies on silicosis, which may have important practical applications. He was interested in and helped every young man who appealed to him for an opportunity to investigate medical problems.

A detailed account of Banting's contributions to medicine will undoubtedly appear in many medical journals, but the time has not yet arrived when the significance of these findings, which his genius for organization has made possible, can be accurately appraised. He was always interested in the organization of medical research, but from the time he realized that war was imminent, he threw himself heart and soul into this work and so fired the imagination of his colleagues and non-medical friends in all parts of Canada that the torch is sure to burn brightly and to illuminate our general war effort. The present activity in the field of aviation medicine in Canada is due largely to him. It is possible that this, his last work, will rank among his greatest achievements.

Banting deeply appreciated the whole-hearted co-operation unstintingly given by many of our American medical colleagues since the outbreak of the present war. He had many personal friends in the United States and often discussed the first American cases which were treated with insulin in Toronto and the physicians with whom this work brought him so closely in contact.

In spite of his great modesty and somewhat shy disposition he had a great capacity for friendship. His activities on behalf of the National Research Council of Canada and his supreme effort to organize medical research for war purposes made him a host of new friends and admirers, but he was always loyal to his medical classmates of 1917 and to those whose advice and help he had received in his earlier struggles.

Banting was true to his philosophy of life which he, at one time, described in the following simple and effective words:

> *It is not within the power of the properly constructed human mind to be satisfied. Progress would cease if this were the case. The greatest joy in life is to accomplish. It is the getting, not the having. It is the giving, not the keeping.*
>
> *I am a firm believer in the theory that you can do or be anything that you wish in this world, within reason, if you are prepared to make the sacrifices, think and work hard enough and long enough.*

C. H. Best

University of Toronto

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TO THOSE STUDENTS OF ANATOMY WHO HAVE SMALL LATIN AND LESS GREEK

By J. C. B. GRANT, M.B., F.R.C.S. (Edin.)
Professor of Anatomy, University of Toronto

To those students of Anatomy who have "small Latin, and less Greek," consolation and courage, for in this respect you rank with William Shakespeare. Nor is it too late to achieve much regarding this shortcoming in your education, and Anatomy may hold the door; at least, it will allow you, by a reversal of the usual school procedure, to acquire a Latin and Greek vocabulary through anatomical terms and thereby to enhance your appreciation of the English language. Need it be said that it is not with a view to affording a vocabulary of anatomical and medical terms that courses in the classical languages are offered in our schools.

Our anatomical tradition takes us back beyond the Romans to the Greeks, back to the third century B.C., back to Alexandria on the delta of the Nile founded by and called after Alexander the Great, Aristotle's royal pupil. To Aristotle we owe the word *enteron* for intestine; from it are derived words such as meso-enteron or mesentery, dysentery, and enteric (or typhoid) fever. To an anatomist at Alexandria we owe the word *duodenum* for the first part of the enteron. This he observed to be as long as twelve fingers are broad, hence in Greek he called it the do-deka-daktulos (2-10-fingers), later to be corrupted into duodenum, duo-decim being Latin for 2-10, that is, for twelve. Evidently, then, to decimate is not to annihilate or exterminate, as certain newspaper reporters would have us think, but to destroy one in every ten.

Twelve islands, of which Rhodes is the largest, lie at the eastern end of the Mediterranean and are known as the Dodecanese Islands, but if you prefer to call them the Duodenal Islands, there is precedent to support you. The island of Rhodes means the island of roses. Rhododendron is Greek for a rose tree, dendron being a tree. Hence, the dendron or *dendrite* of a nerve cell is a process having the appearance of a tree, and the appearance of a dendrite ulcer of the cornea can readily be imagined.

Diminutive Terms

The Fascists of Italy have appropriated as their badge the fasces which was the emblem of authority in ancient Rome. This was an axe embedded in a bundle of rods of elm or birch, tied securely together, and having the obvious and forceful significance that the people (the rods or branches) were subject to authority (the axe). Fascis is Latin for a bundle. The diminutive term, *fasciculus*, is Latin for a small bundle and it is commonly applied to a small bundle of fibres, particularly of nerves and muscles. The Latin terminations —ulus, —ula, —ulum, which in English become —le, are frequently recurring in anatomical writings and suggest things diminutive or small. Even the advertisements in the daily papers refer to the *cuticle* (Latin cutis = skin) or film of skin which, unless restrained, spreads over the *lunule* (L. luna = moon) or small moon near the root of the fingernail. The *clavicle*, which is the first bone you encountered, is probably so-called from its resemblance to the piece of stick used in olden days as a key to raise the primitive latch of a door (L. clavis = a key; clavicula = a small key). The word for a blood cell or blood *corpuscle* is derived from Latin corpus = a body; corpusculus = a small body; and the words corpse for a dead body and corps, as in O.T.C., have the same derivation. A *glomerulus* of the kidney resembles a small ball (L. glomus = a ball). The word *tubercle* (L. tuber = a swelling; dim. tuberculum) is another such word; a potato is a tuber; a small swelling on a bone is a tubercle; and in the pathological condition known as tuberculosis small round inflammatory swellings, called tubercles, are scattered

through the lungs and other organs. The chambers of the heart, *auricle* and *ventricle*, are diminutive terms (L. auris = ear; dim. auricula. L. venter = belly or stomach; dim. ventriculus). Other such words are: capsa = a box, dim. capsula = a *capsule;* lobus = a lobe, dim. lobulus = a *lobule*; malleus = a hammer, dim. *malleolus;* navis = a ship, dim. adjective *navicular*, hence the navy and the navicular bone.

Doubtless, you could extend this list; you could add the word *muscle* (L. mus = a mouse; musculus = a little mouse) meaning a small male mouse, because it would be muscula if it were a female mouse, and musculum if it were neuter. Hence the names of all muscles are masculine, e.g., musculus trapezius, musculus deltoideus, musculus gluteus maximus, musculus rectus abdominis or straight muscle of the abdomen, and musculus pectoralis major (minor) or the larger (smaller) muscle of the chest —had it been neuter it would be majus (minus). For the sake of brevity, however, it is customary to omit the noun (musculus) while retaining the qualifying adjective (e.g., trapezius, deltoideus, rectus, biceps).

Tunica (Latin for a tunic or coat) is an example of a feminine word commonly omitted, e.g., Tunica cornea = the horny coat; T. sclera = the tough coat; T. conjunctiva = the conjoined or united coat; T. media = the middle coat; T. submucosa = the submucous coat.

Intestinum (Latin for the bowel or guts) is a neuter word commonly omitted, e.g., intestinum duodenum (discussed above); i. jejunum = the empty intestine; i. ileum = the twisted intestine; i. rectum = the straight intestine. The rectum takes the shape of the sacrum and, therefore, is straight in lower animals, but in man who walks erect the sacrum is curved and so is the rectum.

Suffixes

Forma is Latin and *Eidos* is Greek for shaped-like or resembling, and they are employed as suffixes thus: —form and —oid. Examples are cribriform = like a sieve; cuneiform = like a wedge; falciform = like a sickle; lentiform = like a lentil or lens; piriform = like a pear; pisiform = like a pea; sacciform = like a sac or bag; and vermiform = like a worm. Clinoid = like a bed, hence clinical medicine and clinics; conoid = like a cone; coracoid = like a crow (crow's beak); cricoid = like a ring; cuboid = like a cube; ethmoid = like a sieve; hyoid = U-oid or like the letter U; mastoid = like a breast; sigmoid = like the letter sigma or S; sphenoid = like a wedge; thyroid = like a shield; trapezoid = like the trapezium; typhoid fever = like typhus fever; and tabloid = like a tablet, and is a proprietary name for the tablets of a certain English Chemical Firm.

A Few Recurring Root Words

Akros (Gk.) = a point or summit; e.g., acromion (akros & omos = point and shoulder), acromegaly, acrocephaly, acropolis, acrobat. *Didumos* (Gk.) = a twin, hence the testis; e.g., epididymis = the organ "on the testis" and paradidymis = the organ "beside the testis". *Ducere* (ductum L.) = to lead; e.g., abduct (ab = from); adduct (ad = to); aqueduct (aqua = water); Il duce = the leader. *Fornix* (Gk.) = an arch, e.g., of the conjunctiva, vagina, and brain. *Genesis* (Gk.) = the first book in the Bible = beginning, origin, creation; c.f., gene, genetics, eugenics, genital, generation. *Lemma* (Gk.) = a husk; e.g., neurilemma, sarcolemma (sarx = flesh, hence sarcomere, sarcoma, sarcophagus). *Phusis* (Gk.) = growth; e.g., diaphysis or the part growing between the two ends of a bone; symphysis or growth together, as in symphysis pubis and symphysis menti; epiphysis or growth upon; and hypophysis cerebri or growth under the brain, also called the pituitary gland. *Skopein* (Gk.) = to look at or examine; e.g., microscope (mikros = small); laryngoscope; ophthalmoscope; periscope (peri = around); stethoscope (stethos = the chest); and telescope (tele = at a distance, far off).

Prefixes

Circum is Latin and *peri* is Greek for round about, around. Circumference is Latin for periphery, which is Greek. Examples: circumduct (lead or conduct

around), circumflex (bend around), circumvallate (rampart around), and circumcise (cut around). Pericardium (around the heart), perichondrium (around cartilage), periosteum (around bone), perineal (around the "anus and urethra"), and peritoneum (stretched around), and periscope (look around).

Dia (Gk. = between, through, across; e.g., diaphragm = a partition between; diameter = a measurement across; diarrhoea = to flow through; *diabetes* = to pass through or syphon; and di(a)encephalon = the in-between-brain.

Epi (Gk.) = upon; e.g., epidermis = "upon the skin"; *epiphysis* = growth upon; *epiglottis; epitympanum;* and *epidemic* (demos = the people—hence democracy) = a disease descending on the people.

Hypo (Gk.) and *Sub* (L.) = under, below; hence hypodermic and subcutaneous, dermis being Greek for skin, and cutis being Latin.

Hyper (Gk.) and *Supra* (L.) = above, over, excessive; hence hypernephric and suprarenal, nephros being Greek for kidney, and ren being Latin.

Para (Gk.) = beside, alongside; e.g., paracolic fossa, paranasal sinuses, parathyroid gland, paradidymis, parovarium. parotid (ous, otikos = the ear) gland, and parallel lines.

Pro (L. and Gk.) = before; e.g., prostate = the gland standing before the bladder; pronephros = the front (cervical) kidney; projection = thrown forwards; protuberance = a forward tuber or swelling; protract = to pull forwards; and prognosis = known beforehand.

Meta (Gk.) = beyond, after, over; e.g., metacarpals and metatarsals = the bones beyond the carpals and tarsals; metopic = beyond the optic or eye region; metanephros = the after or hind kidney; and metabolism and metaphor.

Syn (Gk.) = with, together with; e.g., synchondrosis = united with cartilage; synbiosis (bios = life; hence biology) = living together; synchronise = timed together (chronos = time, ch., chronic); synapse = joined together (aptein = to join); synopsis (opsis = a view) = a general view; symposium (potes = a drinker) = a drinking party; and synagogue.

Tele (Gk.); distant, end; e.g., telencephalon = the end brain; telescope (skopein = to see); telephone (phone = the voice); and teleology.

Trans (L.) = across, through; e.g., transpyloric plane; transfuse (fusus = poured); transverse (versus = turned) = turned across the long axis; translucent (lucere = to shine); transparent (parere = to appear); transpire (spirare = to breathe) and transude (sudare = to sweat).

A v. A: A in Greek = not, the negative; e.g., aseptic (septos = decay) means not-decayed; and atrophic (trophe = nourishment) means not-nourished. A or Ab in Latin = from, away from; e.g., to avert (vertere = to turn) means to turn away from; and to abduct (ducere = to lead) means to lead away from. Non is Latin for not, the negative; e.g., non-vascular (vas = a vessel; vasculum = a small vessel), vas being a Latin word, avascular would mean "from vascular".

Dis v. Dys: Dis (L.) apart, asunder; e.g., dissect (secare = to cut) = to cut apart; dislocate (locare = to place) = to put out of place; and disseminate (seminare = to sow; semen = seed) = to scatter seed. Dys (Gk.) = difficult, painful, not good; e.g., dyspepsia (pepsis = digestion); dystrophy (trophe = nourishment; c.f., atrophy, hypertrophy); and dysentery (enteron = intestine).

Ante v. Anti: Ante (L.) = before, in front of; e.g., anterior, anteflexed, antebrachial, ante-"room", and ante-"dated". Anti (Gk.) = opposite, against; e.g., antihelix, antitoxin, antidote, and antagonist.

Coli v. Colli: Coli (L.) = of the colon. Colli (L.) = of the neck.

Ileum v. Ilium (L.) are neuter adjectives. Intestinum ileum = the twisted intestine; os ilium = the flank bone.

Inter v. Intra (L.) are prepositions. Inter = between; intra = inside, within; e.g., interlobular = between lobes; intralobular = inside lobes.

Os v. Os: Os (oris, L.) masculine = a mouth. Os (ossis, L.) neuter = a bone.

Perineal v. Peroneal are (Gk.) adjectives. Perineal = around the "anus and urethra". Peroneal = fibular; perone being a fibula or a pin.

Of all anatomical terms, certainly of all anatomical eponyms, you would

imagine *tendo Achillis* to be the most ancient, dating back to Homeric days and the siege of Troy, but Dr. J. Harold Couch, in a short paper published in the Canadian Medical Journal, page 668, June, 1936, dispells this notion and attributes the term to Philip Verheyen, Professor of Anatomy in the University of Louvain, who introduced it into our nomenclature in 1693, as *chorda Achillis*, later to be modified to tendo Achillis, and now commonly called the *tendo calcaneus*. Indeed, with the advance of knowledge, it becomes necessary to make and to introduce new names, hence most anatomical terms are post-renaissance words.

If you have doubt as to the correctness of the interpretation given here to any word, your appeal is to the dictionary—to a Latin and a Greek dictionary, the Shorter Oxford English Dictionary and a medical dictionary; and their value diminishes in this order.

The Attributes of the Heart

"Since many ages the heart has had the report, not only to be the principle, or the beginning of life, but of the whole body, in which the soul has taken up its dwelling houfe, and from which as from a fountain all the vital faculties and fpirits do flow. Plato calls this the feat of the Irafcible Soul; Aristotle calls it the feat of the Vegetative, Senfible, and Ratiocinative Soul. Befides it vaunts itself to be the ftore houfe of our moifture, the fire and nutriture of our native heat, the Sun of our body, by whofe influx all the bowels are warm and refrefhed: moreover here they fay that artificial fire of Zeno is contain'd, here the Divine and celeftial heat is preferv'd, which the Poets feign Prometheus to have stole from heaven, that he might put in life in man; Therefore it is called the firft moving, and the firft mover, and the firft if not the only ftore-houfe for making of blood."

The Discourse of James DeBack
London—Printed by Francis Leach, 1653.

ACUTE HAEMATOGENOUS OSTEOMYELITIS IN CHILDREN

By F. R. WILKINSON, M.D.

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Definition

Acute osteomyelitis is an infective inflammation of bone. It is "a boil in a bone".

The bone may be infected:

1. By way of the blood stream.
2. By direct extension from the soft tissues.
3. By open wounds.

Only the first of these will be discussed.

Anatomy

Bone is elastic and strong—almost as strong as cast iron and about twice the strength of oak. Except for the enamel of the teeth it is the hardest and most enduring tissue in the body.

The *periosteum* is a fibrous membrane adherent to the surface of bone except where cartilage covers the articular surface. It is firmly attached to the epiphyseal line and is not continuous with the periosteum covering the epiphysis. The *endosteum* is a vascular areolar membrane lining the medullary cavity.

The *cortex* is composed of a series of Haversian systems consisting of an Haversian canal about 0.05 cm. in diameter traversed by blood vessels and lymphatics and surrounded by concentric lamellae of bony tissue. Between the lamellae are minute lacunae, containing bone cells, which communicate by very fine channels, called canaliculi, radiating like the spokes of a wheel. Depending upon the age of the patient this compact bone varies from one eighth to one quarter of an inch in thickness in the mid-shaft of the long bones and tapers off gradually to tissue paper thinness at the epiphysis.

Cancellous bone is found at the ends and in the interior of the long bones. It is much looser architecturally than compact bone though the structural units are similar.

The *medulla* fills the medullary or central canal and also occupies the interstices in the cancellous tissue. It is either yellow or red and consists of connective tissue, fat, blood vessels, myelocytes and erythroblasts.

The *epiphyseal cartilage* is a layer of cartilage cells separating, in the growing bone, the diaphysis or shaft from the epiphysis. Here active growth takes place.

The *Blood supply* of long bones is derived from two sources:

1. The nutrient artery which perforates the cortex to reach the medulla.
2. Vessels from the periosteum which anastomose with branches of the nutrient artery.

In children the epiphysis is supplied by vessels from the capsule of the joint.

Etiology

1. Predisposing Causes

1. Acute haematogenous osteomyelitis presupposes a primary focus elsewhere in the body. In about one third of the cases due to staphlococci there is a demonstrable skin lesion such as a furuncle, an infected wound or abrasion, blister, burn, impetigo, chicken pox or umbilical sepsis. Streptococci may enter the blood stream from infected teeth, tonsils or sinuses.
2. *Trauma* is an important predisposing factor. Just how important it is there is no general agreement. Schmidt found a history of trauma in 66 per cent of the cases; Pfenningsdof in 41 per cent, and Willinsky in only 5 per cent.

3. *Age*: The disease occurs most commonly between the ages of 2 and 10 years.
4. *Sex*: Boys are affected more commonly than girls in the proportion of about 3 to 1.
5. *Geographical distribution* and seasonal variation are probably not important.

2. Exciting Causes

Staphlococci, usually the haemolytic staphlococcus aureus is the cause in at least 90 per cent of cases. Streptococcus haemolyticus is found in 3 to 5 per cent, and, less frequently, the pneumococcus, typhoid bacillus, colon bacillus and gonococcus.

Localizing Influences

1. The infection begins in the metaphysis close to the epiphyseal line. Here the arteries are end arteries and loops and sinuses are formed wherein the blood flows sluggishly. This favours the lodgement of bacteria.
2. Phagocytosis is less active in the metaphysis than in the medulla.

Pathology

The late Professor C. L. Starr has shown conclusively by animal experiments, X-rays, and autopsy specimens that the infection spreads from the focus in the metaphysis along the line of the epiphysis through the thin cortex to the periosteum. Here a subperiosteal abscess extends rapidly, stripping up the periosteum and depriving still further the bone of blood. The medulla is then invaded from the subperiosteal abscess through the Haversian canals—*not* by direct extension from the metaphysis. Necrosis of bone follows. The bone dies because it is poisoned by toxins, made anaemic by pressure, and deprived of its blood supply by thrombosis and by stripping of the periosteum. The extent of necrosis depends largely upon how much stripping of periosteum there has been.

Dead bone is called *sequestrum*. Rarely the whole shaft may die forming the so-called *tubular sequestrum*.

Later on subperiosteal new bone, the *involucrum*, is formed.

Holes in this new bone, due to perforations in the periosteum, are called *cloacae*.

Vascular granulation tissue forms on the living bone and in time produces separation of the sequestrum. The separation requires from 2 weeks to 6 months, depending upon the density and thickness of the bone involved.

The chief causes for the persistence of sinuses in this disease are the presence of sequestra and of a deep cavity with rigid walls.

The bones affected in order of frequency are:

Upper tibia
Lower femur
Lower tibia and fibula
Upper humerus
Lower radius
Lower humerus
Upper femur
Crest of ilium.

Signs and Symptoms

The diseases usually begins suddenly. Let us consider a typical case history. 1 week ago, the patient, an 8 year old boy, noted an infected abrasion on his foot. 3 days ago, he twisted his knee and limped for a few hours, then seemed perfectly well. (At this time he was suffering from a symptomless bacteraemia.) 2 days before admission he began to complain of general malaise, headache and chilliness with constant, throbbing pain in the knee. (Now the bacteria have settled in the upper tibial metaphysis and are multiplying there. The boy's symptoms are due to toxaemia and to the local lesion.) On the day before admission the parents observed a swelling below the right knee. The child was unable to sleep that night because of the pain; the swelling increased, and he was brought to hospital the following day.

On admission he looks acutely ill. He

is flushed, restless, apprehensive, with a dry, furred tongue. The right leg is held with the knee flexed. Over the antero-medial aspect of the upper third of the right tibia is a red, warm swelling. This is exquisitely tender; indeed the child screams when it is touched. Fluctuation is not present. It is possible to move the knee very gently without increasing the pain. There is no fluid in the knee joint. His pulse rate is 130; temperature 104°; white blood cell count 25,000. X-rays show soft tissue swelling but no lesion in the bone.

Diagnosis

The diagnosis should be made within 24 hours of the onset and the patient sent to hospital. The results in this serious and crippling disease will improve when the patient consults the doctor early, and when the doctor makes an early diagnosis.

Every case of severe pain near a joint with a point of maximum tenderness over the bone end in a growing child should be considered due to osteomyelitis until proved otherwise.

If accompanied by toxaemia, rapid pulse, high temperature and a high leukocyte count, the diagnosis is reasonably sure.

X-rays are of no value at this stage and should not lull one into a false sense of security. The earliest X-ray change is usually seen about 7 to 10 days after onset and consists in a reduction in density due to bone obsorption. Several days later areas of destruction and of superiosteal new bone formation are visible.

Differential Diagnosis

In *Acute Suppurative Arthritis* the signs are primarily in the joint, not at the metaphysis. There is an early effusion, muscle spasm is more marked, and limitation of motion greater. Aspiration of the joint and examination of the fluid by direct smear and culture will settle the question.

Acute Rheumatic Fever may cause confusion but the onset is more gradual, the pain less severe, and usually more than one joint is involved. Valuable time should not be wasted by giving salicylates as a therapeutic test.

In *Erysipelas* the skin is red and shiny with a raised edge.

Cellulitis may suggest osteomyelitis but the swelling and redness appear earlier. The pain is not so intense and the general toxaemia is less.

Infantile Scurvy causes haemorrhage beneath the periosteum resulting in pain, fever and swelling with marked periosteal new bone formation.

Complications

The most common complication is *Acute Suppurative Arthritis* which may arise either by direct extension to the joint or haematogenously. It is more frequently due to direct extension.

Tenosynovitis may also arise by direct extension.

Secondary foci in other bones are said to be present in about one-third of the cases in children.

Pathological fracture may occur.

Amyloid disease sometimes supervenes in the long standing suppurative cases.

Carcinoma is a late complication and develops in cases where the sinus has persisted for a long time.

Prognosis

Osteomyelitis in children is a dangerous disease. Kennedy reported a mortality of 34 per cent in 309 cases; Lloyd 32 per cent in 39 cases; Krasnobajev 22.4 per cent in 428 cases; Williams 19.7 per cent in 91 cases. At the Hospital for Sick Children, from 1923 to 1936, there were 674 cases with 140 deaths, a mortality of 20.77 per cent. In the past 15 months, from January, 1940 to March, 1941, inclusive, there have been 33 cases with 2 deaths, a mortality of 6 per cent.

Straphlococcal infection is the most serious type both as regards life and bone destruction.

The nearer the trunk the affected bone

and the younger the child, the graver is the prognosis.

6 to 18 months usually elapse before a temporary "cure" is obtained.

The disease may become chronic and last for decades. Recurrences and relapses are common. Osteomyelitis, like tuberculosis, is arrested—not cured.

Treatment

The treatment of Acute Haematogenous Osteomyelitis at the Hospital for Sick Children is as follows:

1. General

On admission the child is put to bed; given a sedative; fluids, glucose and saline intravenously, if necessary, and if necessary a blood transfusion.

Sulphathiazole, gr. iss to gr. ii per pound of body weight in 24 hours is exhibited. One half the daily dose is given at once, then divided doses every 3 hours. For instance, if it is decided to give the child gr. CXX a day the order is written:

Sulphathizole gr. LX Stat.
Then gr. XV q. 3 h. x 8

If the patient cannot take the tablets by mouth the soluble sodium sulphathiazole is given intravenously in the same dosage.

Operation is done:

1. When the patient's general condition is satisfactory.
2. When the level of sulphathiazole in the blood is 5 mgms. per cent or higher.
3. When we are reasonably sure that pus is present.

These conditions are usually fulfilled within 24 hours of admission.

It is felt that children with osteomyelitis die of an overwhelming septicaemia; they do not die because one neglects to drain an abscess in the bone. We believe that immediate operation, before general measures to combat the septicaemia have been instituted, is contraindicated. Where there is pus it should be let out but not at the expense of the patient's life.

2. Local—The Operation

Under a general anaesthetic, and without a tourniquet, an incision is made over the affected bone. The periosteum is incised, pus evacuated, and two drill holes made through the cortex into the metaphysis. The wound is packed loosely with gauze impregnated with acriflavine in glycerine or some other greasy material. It is left widely open. A dry dressing is applied and the extremity placed in plaster or in tractiom. The operation takes only a few moments and is not a shocking procedure.

The dressing is not changed until the end of a month, unless the smell becomes unbearable, or unless there is concern about spread of infection. The packing is removed at the end of a month and the wound is not repacked. At the end of the first week the dose of sulphathiazole is cut in half. The drug is continued for at least 4 weeks.

Conclusions

It is too soon to draw definite conclusions from the small series of 33 cases but the results of this plan of treatment are encouraging and the following observations suggestive:

1. The mortality is 6 per cent.
2. The clinical course of these patients is better than it used to be.
3. It is thought that there has been less bone destruction.
4. As yet there has been no secondary focus in another bone.

The work on osteomyelitis is aided by a grant from the Nemours Foundation, Wilmington, Delaware, and is being carried out at the Hospital for Sick Children under the direction of Dr. D. E. Robertson, Surgeon-in-Chief, and at the Banting Institute.

DIABETES

By R. E. HAIST, M.A., M.D., PH.D.
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Half a million people in the United States are suffering from diabetes and even in our own province of Ontario, diabetes ranks among the 10 chief causes of death. It will be evident that diabetes is a medical problem of major importance. It will be clear too that a knowledge of the factors influencing the development and course of the condition is a necessary prelude to prevention or cure.

Von Mering and Minkowski in 1889 were the first to show that diabetes can be produced in dogs by removal of the pancreas. Since that time, the depancreatized dog has been extensively studied. For brevity we shall list some of the findings in these animals. They become emaciated though they are fed well, and exhibit: excessive hunger and thirst; polyuria; hyperglycaemia; glucosuria; ketonaemia and ketonuria; glucosuria; nitrogen excretion; increased new formation of sugar from other substances (gluconeogenesis); decreased ability to use carbohydrate and decreased ability to form glycogen. They usually die in less than 3 weeks if untreated.

It will be evident that the depancreatized animal shows profound changes in carbohydrate metabolism and also in the metabolism of protein and fat. The experiments in which the pancreas was removed indicated that normally something was provided by the pancreas which was necessary to prevent the occurrence of diabetes. It was not until 1922 that a potent, non-toxic extract of pancreas was prepared by Dr. Banting and Dr. Best at this University. By the use of this extract they were able to alleviate the diabetes of depancreatized dogs and the diabetes of hospital patients.

The effect of insulin in the diabetic is to relieve the diabetes and what more need be said? This does not hold entirely for the depancreatized dog since this animal lacks the other pancreatic secretions as well. Such dogs require raw pancreas or lecithin or choline in addition to the insulin.

Insulin is produced in the pancreas and there is no evidence for any extrapancreatic source. The β cells of the islets of Langerhans seem to be the essential endocrine structures. Their importance is indicated by some work done in collaboration with Dr. Best, Dr. Campbell and Dr. Ham in which it was shown that the insulin content of the pancreas under different experimental conditions is closely related to the degree of β cell granulation. When the β cell granulation is normal the pancreas has a normal concentration of insulin and when the β cell granulation is reduced the insulin concentration in the pancreas is reduced also. It would appear from what has been said that diabetes results from a deficiency of a particular hormone, insulin, manufactured by the β cells of the pancreatic islets.

For a while many people were sure that the pancreas alone was responsible for diabetes and the problem seemed to be satisfactorily settled. However, *no problem is settled if people continue to observe.* The unsettling observation relating to this subject was one made by Houssay and his associates, namely, that diabetes caused by removal of the pancreas was ameliorated by taking out the pituitary gland. More recently Long and Lukens have shown that removal of the adrenals will relieve the diabetes due to pancreatectomy. It was found too that certain crude extracts of the anterior pituitary gland when injected into the hypophysectomized-depancreatized animals brought a return of the diabetic state. These extracts were also found to give rise to a diabetic state when injected into normal animals, i.e., animals with intact pacreas. No one has yet produced typical diabetes by the injection of adrenal cortical substances.

As a result of these experiments, no longer can one say that the *absence* of β

cells alone is responsible entirely for the diabetic picture. It would appear that the *presence* of the pituitary and certain other endocrine glands is involved. There are then two antagonistic systems, one necessary to prevent diabetes and one helping to cause diabetes. It is obvious that diabetongenic and antidiabetic factors only show their influence by their effect upon some other structure or structures important in the new production, storage, liberation or use of sugar. The structures acted upon seem to be liver and peripheral tissues, chief of which is skeletal muscle. It is important to remember that the activity of these structures will be affected by diet as well as by the factors mentioned. It is probably also true that the activity of liver and peripheral tissues, by influencing blood sugar or blood insulin level, affects the activity of those structures responsible for the diabetongenic and antidiabetic effects (e.g., pituitary, adrenals and pancreas).

As yet we do not know whether or not there is a continuously acting but compensated "diabetogenic" influence in the normal animal. Regardless of what the condition in the normal may be we still may consider that, in the diabetic, there is some alteration in the balance between diabetogenic and antidiabetic factors. This will be brought out more clearly by a review of the two methods for producing experimental diabetes in which some pancreatic tissue remains, namely, (1) extensive partial pancreatectomy, (2) injection of certain anterior pituitary extracts.

The removal of all or nearly all the pancreas had been shown to cause diabetes. Then Homans and Allen demonstrated that by leaving a very small bit of pancreas in the animal the changes in the islet cells could be observed. They found that removal of a large portion of the pancreas in dogs led to diabetes, accompanied by degranulation, and finally hydropic degeneration in the β cells of the islets of Langerhans. Allen showed that the intensity of the diabetes paralleled the severity of the islet cell changes. He developed the thesis that the degeneration resulted from exhaustion of the islet cells through overwork. Some cast doubt on Allen's conclusions on the basis that the material in the hydropic cells might be insulin.

We have been able to demonstrate that when enough pancreas is left in a dog that it does not become diabetic, the insulin concentration in the pancreatic remnant is normal, whereas if sufficient pancreas is removed so that diabetes results, the insulin concentration in the remnant falls to very low levels. Our findings support Allen's conclusions that the effect of extensive partial pancreatectomy on the islet cells is the result of overwork, and certainly indicate that the material in the vacuoles is not physiologically active insulin.

Houssay, Evans and others demonstrated that the injection of anterior

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EXTENSIVE PARTIAL PANCREATECTOMY LEADS TO:

1. Diabetes
2. Degranulation of β cells of islets of Langerhans
3. Hydropic degeneration of β cells of islets
4. Great reduction in insulin concentration in pancreas remnant

Fasting or insulin administration reduces 1, 2 and 3 (and presumably 4, though it has not been tried).

DAILY INJECTION OF ANTERIOR PITUITARY EXTRACT LEADS TO:

1. Diabetes
2. Degranulation of β islet cells
3. Hydropic degeneration of β islet cells
4. Great reduction in insulin concentration in pancreas.

Adequate administration of protamine zinc insulin, fasting or fat-feeding tend to prevent 1, 2, 3 and 4.

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pituitary extracts gave rise to a diabetic state in dogs. Our group here—Dr. Best, Dr. Campbell, Dr. Ham and myself—have demonstrated that the daily injection of certain extracts of anterior pituitary gland leads to a diabetic state associated with a progressive reduction in the insulin content of the pancreas and a progressive degranulation of the β cells of the islets of Langerhans followed by hydropic degeneration of those cells. The picture is similar to that found in the partially depancreatized animals and is thought to be due to the same cause, namely, overwork of the β cells of the islets with consequent exhaustion. Young had shown that a sufficiently long course of injections of anterior pituitary extract led to a diabetic state, associated with islet changes which persisted indefinitely after the injections were discontinued, i.e., a so-called permanent diabetes. The exhaustive overwork, if it continues long enough, finally leads to a permanent reduction in functioning β cells and in insulin content and the diabetic state persists even though the anterior pituitary extract is no longer administered. In the permanently diabetic dog, then, it would appear that diminished islet function is a factor of major importance. Hence it it very much like the depancreatized animal but differs in this respect, that it still has its α cells and external pancreatic secretion.

As a necessary preparation for the problem of prevention of these diabetic effects we should consider the result of changes in diet in normal animals. It was thought that from the study of dietary changes we might be able to learn something regarding the factors which governed the activity of the pancreatic islets. One of the first discoveries made with Dr. Best and Dr. Ridout was that fasting or the feeding of fat led to a definite reduction in the insulin content of the rat pancreas. Sugar tended to prevent this reduction.

When, after a starvation period of 1 week, the animals were given sugar or a balanced diet, the insulin content of the pancreas rose from the low fasting level and returned to normal. With the sugar alone, the normal values were not maintained. Lately we have found that, after starvation, feeding protein gives values much the same as for sugar. It seems likely that the effect of the protein is due to its ability to supply sugar, though this is not yet clearly established. Fat has no effect in restoring the insulin content. From these experiments it seemed evident that fasting and fat-feeding lead to a reduction in the insulin content of the rat pancreas, largely through the absence of carbohydrate or materials that may form carbohydrate. Other factors may be involved but this seemed to be the important one.

As far as could be determined, the effect of fat was similar to the effect of starvation but if anything, more definite. Since the fat-fed animals got fatty livers it was possible that damaged liver function was responsible for the change in the fat-fed animals. If this were true, then starvation and fat-feeding might really have different effects. However, recently we have found that fat-fed animals given large amounts of choline to hinder the liver fat deposition still showed the striking reduction in the insulin content of the pancreas, even though the liver fat remained the same as in the controls.

While fasting and fat-feeding led to a reduction in the insulin content of the pancreas it was not yet clear whether this was due to a decreased production of insulin and a resting of islet cells or whether it was due, as in the case of the partially depancreatized dogs and dogs given anterior pituitary extract, to an increased activity and exhaustion of islet cells. The "resting" hypothesis seemed more logical. It was thought that if large doses of protamine zinc insulin were given some light on this question might be obtained.

The daily administration of adequate amounts of protamine zinc insulin led to a reduction in the insulin content of the pancreas. This reduction could hardly be ascribed to overwork and it seemed logical to conclude that it resulted from a decreased production of insulin by the islet cells.

When daily injections of protamine

zinc insulin were given to fasted and fat-fed rats it was found that the effect of fasting and fat feeding was enhanced. Insulin did not prevent the fall in insulin content due to fasting and fat-feeding; it augumented it. Consequently it seemed necessary to conclude that insulin administration, fasting and fat-feeding had similar effects and since insulin administration rested islet cells, therefore the other procedures must also tend to rest the islet cells. The absence of histological evidence of degenerative changes in the pancreas of these animals also argued for a resting effect rather than overwork.

From the work on rats it was concluded that insulin administration, fasting and fat-feeding tended to rest islet cells. Now, if it were true that fasting, fat-feeding and insulin administration rested the islets, then these procedures should prevent overwork of islet cells and prevent experimental diabetes in the dog.

As far as the partially depancreatized animals were concerned the literature confirmed our expectations. Allen had found that fasting alleviated the diabetes due to partial pancreatectomy and restored the islet cells to a more normal state. Copp and Barclay and Bowie found that adequate insulin administration had a similar effect in restoring islet cells. There remained but to try the effect of these procedures in the animals receiving anterior pituitary extract. Dogs receiving daily injections of anterior pituitary extract show a great reduction in the insulin content of the pancreas and hydropic degeneration of β cells of the islets, as mentioned previously. In some experiments done in association with Dr. Best, Dr. Campbell and Dr. Ham, it was found that when daily injections of protamine zinc insulin are given to dogs along with the injections of anterior pituitary extract, the fall in insulin content is hindered and the degeneration of the islet cells is, to a large extent, prevented. Fat-feeding and fasting in dogs given anterior pituitary extract have similar effects. The fall in insulin content of the pancreas is to a large extent, prevented and the degenerative changes are not evident.

It seems clear then that in the dog the effect of insulin administration, fasting and fat-feeding act in a manner opposite to the effect of partial pancreatectomy and of anterior pituitary extract administration. This further substantiates the contention that these procedures of fasting, fat-feeding and insulin administration rest the pancreatic islets. It seems evident that the factors which rest the islet cells in the rat tend to prevent the onset of experimental diabetes in the dog.

We are interested to know how fasting, fat-feeding and insulin administration bring about their effect. We may conclude that fasting, fat-feeding and insulin administration rest the pancreatic islets by reducing the need for endogenous insulin. From what we have said previously it will be evident that this effect might be brought about either by reducing the activity of those structures on the diabetogenic side of the balance, such as the pituitary or adrenal, or by directly affecting the liver and peripheral tissues, i.e., those tissues involved in the new formation, liberation, storage or utilization of sugar.

In rats, the effect of fat-feeding on the insulin content of the pancreas can still be obtained in the absence of the pituitary or adrenal glands. It would appear that the pancreatic islets can alter their function acording to the need for insulin in the absence of the pituitary and adrenal glands. It seems probable therefore that the effect of fat-feeding and presumably the effect of fasting also is brought about by an influence on liver and peripheral tissues, rather than through some other endocrine. The evidence at present seems to indicate that the absence of carbohydrate in the diet affects the activity of liver and peripheral tissues, presumably reducing the need for insulin and resulting in changes in blood sugar or blood insulin levels which decrease the stimulation of the islet cells. In the diabetic we may assume either that these procedures make the tissues less responsive to the diabetogenic influence or else directly reduce these influences.

Further work is required to ascertain what are the important processes in liver and peripheral tissues which are affected

by carbohydrate or protein intake and which require insulin.

Summary

Summarizing some of the points discussed in this paper, we may say that:

(1) Fasting, fat-feeding and insulin administration lead to a reduction in the insulin content of the pancreas.

(2) The effect of fasting and fat-feeding is enhanced by the administration of insulin.

(3) The effect of partial pancreatectomy and of anterior pituitary extract administration is hindered by insulin administration.

Scraps of Medical Folk-Lore

"The domestic treatment of disease among our poor consists, chiefly of charms and ceremonies. In many cases also we may notice remnants of old medical creeds, such as the doctrine of signatures, and the notion of sympathies and antipathies between separate and dissimilar bodies. In the cure for haemorrhage, preference is shown to medicines of a bright red colour. The fisherman whose hand has been pierced by a hook is very careful to preserve the hook from rusting during the healing of the wound.

If an infant is suffering from the thrush, it is taken, fasting, on three following mornings, to have his mouth blown into by "a person who never knew his father." If afflicted with whooping-cough, it is fed with the bread and butter of a family, the heads of which bear the names John and Joan; or if a piebald horse is to be found in the country, the child is passed thrice under its belly. With us, boils are cured by creeping on the hands and knees beneath a bramble which has grown into the soil at both ends. Children affected with hernia, are still passed through a slit in an ash sapling before sunrise, fasting; after which the slit portion are bound up; and as they unite, so the malady is cured."

The History of Polperro,
(A fishing town on the coast of Cornwall)
Jonathan Couch, F.L.S.
Country Doctor, 1852

ANSWER TO PROBLEM IN LAST ISSUE

By W. K. KERR, M.D.
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Pathological Report

At operation a right cerebellar exposure was made and the following pathological picture was revealed as the operation proceeded.

Note: For the sake of clarity the findings at operation and autopsy are presented together.

1. When exposed the lateral three-quarters of the right cerebellar hemisphere was found to be a thin shell not more than 1 cm. thick lying over the firm, smooth, whitish, rather nodular tumour, the size of a small golf-ball, arising from an enlarged jugular foramen. It incorporated the 7th, 8th, 9th, 10th and 11th cranial nerves on the right side and compressed the 5th and 12th nerves. The tumour had produced a marked indentation into the pons and medulla; one nubbin of tumour was the size of a large walnut and had burrowed its way into the structures at the base of the skull.

At autopsy it was found that the 5th left cranial nerve was about four times its normal size, appearing to be involved by a diffuse growth which confined itself to this nerve.

2. On section, the tumour in part consisted of soft, succulent material comparable to an acoustic neuroma; there were some cystic areas and also some very tough areas, almost cartilaginous in nature.

Microscopic examination showed an encapsulated, moderately vascular, highly cellular tumour composed of closely packed, decussating fibroblastic-like cells with occasional contorted, fasciculated whorls, and other areas of hyaline degeneration, the whole resembling a neurofibroma.

Diagnosis

1. Neurofibroma of the 11th right cranial nerve.

2. Neurofibroma of the 5th left cranial nerve.

Discussion

The first symptoms were weakness and atrophy of the right half of the tongue and the right trapezius muscle and slight weakness of the right arm and leg. The 12th nerve, however, was found to be intact although compressed, and this could not have given rise to the tumour. The 8th, 9th and 10th nerves were not involved until years later and must thus have been secondarily involved. This leaves the 11th right cranial nerve which appears to be the only one from which the main tumour could possibly have originated. The early involvement of the 12th and later involvement of the 9th and 10th nerves suggests that the tumour was at first intracranial and later extended out of the skull through the jugular foramen.

The diminished sensation on the left side of the face and the absent corneal reflex on the left side did not fit into the clinical picture of a right side, cerebello-pontine angle tumour and could not be explained until it was found that the patient had a second neurofibroma of the 5th left cranial nerve.

The history and examination gave several leads as to what the nature of the lesion might be. For instance, the attack in 1936 of right-sided head pain, sudden deafness, loss of voice and ataxia would suggest some vascular accident rather than a slow-growing tumour. The ulsating bruit heard in the right occipital region was strongly suggestive of an intracranial aneurysm, but was probably produced by the occipital artery which could be seen pulsating beneath the origin of the atrophied trapezius muscle.

The author is indebted to the Department of Pathology for their kind permission to publish the pathological findings of this case.

VITAMIN K AND PROTHROMBIN

By JAMES G. WATT '41

The story of the accidental discovery of Vitamin K by Dam of Copenhagen is one of the most interesting narratives in present-day medicine. During 1930, Dam was studying the problem of cholesterol metabolism in chicks, and he was attempting to see if they could synthesize cholesterol. He fed the chicks on a synthetic, cholesterol-free diet consisting of casein, starch, salt, mannite, filter-paper and cod-liver oil. He was surprised to find that a large number of the chicks raised on this diet developed a severe haemorrhagic disease. The chicks suffered from subcutaneous, intramuscular, and gastro-intestinal haemorrhages despite the fact that the diet contained adequate amounts of vitamins A, B_1, B_2, C and D. Some of the chicks bled to death from trivial wounds. He decided to investigate this haemorrhagic phenomena further, and after four years of intensive study Schünheyder and he were able to show that this disease was due to the lack of a fat-soluble accessory food factor which they called vitamin K (from the Danish, Koagulatidis vitamin). Adding this accessory food factor to the diet completely cured the disease.

This vitamin was found in hog-liver, hemp seed, certain cereals and vegetables, especially alfalfa hay. In the following two years Schünheyder proved that this haemorrhagic chick disease was characterized by a markedly diminished prothrombin content of the blood. The addition of vitamin K to the diet raised the prothrombin level back to normal and cured the disease.

Soon research workers throughout the world were busy on the possibilities of successfully curing the various haemorrhagic diseases in man with this substance, and Armand Quick made the next contribution. He devised a simple and clinically applicable method for measuring the prothrombin level of the blood.

Soon it was found that the haemorrhagic tendency in certain cases of obstructive jaundice was due to a low prothrombin level of the blood. It was logical to suppose that this new vitamin, which raised the prothrombin level in chicks, might also be effective in elevating the prothrombin level of these jaundiced patients. This proved to be true. It was observed in dogs that if a biliary fistula was produced or the common bile duct ligated so that bile could not enter the intestine, that a marked fall in blood prothrombin occurred. The administration of bile salts or bile to these animals caused a slow return of prothrombin to normal levels, but with alfalfa extracts, known to contain large amounts of vitamin K, were given along with the bile salts, the prothrombin level returned rapidly to normal, requiring only three to five days. Vitamin K concentrates given without bile salts were quite ineffective. Thus we can see that if bile is excluded from the gastro-intestinal tract there is failure to absorb the fat soluble vitamin K which is in some way responsible for a normal prothrombin level in the blood.

Quick caused severe liver damage in dogs by starving them, then administering chloroform anaesthesia for 90 minutes. After five hours the prothrombin level began to fall from normal, and in 48 hours a definite bleeding tendency appeared in some of the dogs. From then on the prothrombin level slowly returned to normal, at a rate comparable to the rate of regeneration of liver cells. This strongly suggests that prothrombin is manufactured in the liver.

It is difficult to produce vitamin K deficiency in man by dietary means, since vitamin K is a constituent of so many foodstuffs, and because the normal bacterial flora of the intestine are able to elaborate it. Recently pure crystalline vitamin K has been prepared from alfalfa and putrifying fish-meal which has a very high potency. Several compounds have been synthesized that have the same physiological properties as vitamin K, and some of these are water-soluble and suitable for oral use without bile salts added.

Others can be given intramuscularly or intravenously. They are very valuable and have therefore supplanted the natural vitamin K for therapeutic purposes.

Briefly summarised, the factors which will cause a prothrombin deficiency are:

(1) *Failure of absorption of vitamin K* from the gastro-intestinal tract. Due to:

(a) No bile reaches the intestine — obstructive jaundice, biliary fistula.

(b) Severe diarrhea—sprue, ulcerative colitis.

(c) Gastro-colic fistula, food containing vitamin K is short-circuited past the small intestine.

(d) Prolonged intestinal obstruction.

(2) *Liver failure*—advanced cirrhosis of the liver, chloroform poisoning, prolonged obstruction of the common bile duct.

To explain how prothrombin deficiency causes a bleeding tendency, it is necessary to digress for awhile and to refresh your memory with the details of the normal physiological process which results in the clotting of the blood. There are two essential processes:

Prothrombin, Thromboplastin Calcium ions, Thrombin

Fibrinogen, Thrombin, Fibrin

Thus there are four substances necessary for the normal clotting of blood; prothrombin, thromboplastin, calcium and fibrinogen. A relative or absolute deficiency of any of these substances might result in delayed clotting time or a failure of the blood to clot. How important to the process is any one of them?

Fibrinogen is a plasma protein and ranges from 0.3 to 0.75 per cent in the normal blood. This wide variation has no effect on the clotting time since fibrinogen is a passive agent. It is in colloidal solution and is converted to an insoluble gel by the action of thrombin. The insoluble fibrin forms fine threads of clot, to which the platelets attach themselves, increasing the adhesiveness of the clot. In haemophilia the prothrombin is normal. It tends to fall in obstructive jaundice and in liver damage since it is made in the liver. There is a low fibrinogen content of the blood in pernicious anaemia, scurvy, pellagra and the leukemias. Three cases are on record of children whose blood was completely incoagulable due to congenital absence of fibrinogen, yet two of them have lived over eight years without evidence of massive haemorrhage. Maybe they are just lucky! This certainly demonstrates well the importance of the vascular mechanism of defense against haemorrhage.

Calcium was believed by Mellanby and Howell to be necessary for the conversion of prothrombin to thrombin. It was thought to act in the ionized form, but Quick has removed all the ionized calcium by oxalate and still obtained clotting. Ionized calcium plays an important role in this lysis of platelets thus releasing thromboplastin. In obstructive jaundice there is no fall in ionized calcium. In the extreme hypocalcaemia of parathyroid deficiency, no haemorrhagic diathesis occurs, even when the serum calcium is as low as 4.6 mg. per cent. *It is not possible to name any bleeding condition due to calcium deficiency.*

Thromboplastin is an enzyme which has the ability to convert prothrombin to thrombin. It is widely distributed in the body as an intracellular substance and is liberated whenever cells are injured. It never occurs free. The platelets contain it in abundant quantities. A decrease in thromboplastin occurs in three ways:

(1) Reduction in the number of platelets as in thrombocytopenic purpura. The platelet reduction also alters capillary permeability, and this is largely responsible for the haemorrhage in thombocytopenic purpura.

(2) Abnormal resistance of platelets to lysis. This occurs in haemophilia. Adding thromboplastin to haemophilic blood causes it to clot with normal rapidity.

(3) The platelets may be deficient in thromboplastin.

Prothrombin is probably a pro-enzyme, and it is the precursor to thrombin, the active coagulating enzyme. It is closely associated with the serum globulin. Thrombin is the resultant of the interaction of prothrombin, ionized calcium and thromboplastin. Heparin and possibly the serum albumin (Quick) act as

anti-thrombins. Studies made on the prothrombin content of the blood show that it has a remarkable constancy in the normal blood and that it is present in large excess above the normal physiological requirements. Quick has evolved a test that measures the clotting time of the blood in the presence of an optimal amount of ionized calcium and an excess of thromboplastin. By once again looking at the equations you will see that the time required for the blood to clot under such conditions is inversely proportional to the prothrombin content of the blood. By measuring the time, one can estimate the prothrombin content of any given sample of blood. The normal prothrombin time is 10 to 15 seconds. The time does not lengthen appreciably until the blood prothrombin falls below 20 per cent of normal.

The Role of Vitamin K in the Treatment of Haemorrhagic Disease

Vitamin K is a specific. It acts in one way. Somehow it stimulates the liver to produce prothrombin, and therefore it will only be of value in bleeding diseases where there is a prothrombin deficiency. It is useless in haemophilia or thrombocytopenic purpura.

Very few patients with obstructive jaundice ever show a bleeding tendency, but one recent report cites fifty cases of fatal bleeding in patients with jaundice. In all the cause of the icterus was complete or incomplete obstruction of the common bile duct. In nearly all there was evidence of hepatic damage. Those cases having carcinoma of the head of the pancreas suffered four times the operative mortality of the cases having obstruction due to stones. This was largely due to the deeper jaundice and greater bleeding tendency because of the more complete obstruction and the greater fall in prothrombin level. There is usually no tendency to bleed in the jaundiced patient before operation, but bleeding occurs soon after, from the wound into the peritoneal cavity, and sometimes spontaneously from mucous membranes. No serious bleeding occurs until the prothrombin level falls below 20 per cent of the normal. What happens is that the prothrombin is not greatly reduced before operation, but the combined effects of the anaesthetic and the using up of prothrombin at the site of operation lower the prothrombin below the danger level, and the prothrombin time becomes markedly lengthened. There is then the danger of haemorrhage.

Treatment might be outlined as follows: give preoperative blood transfusion to supply an immediate reserve of prothrombin, and 5 per cent glucose in saline to increase the glycogen stores of the liver thus protecting it from the depressing effects of the anaesthetic. Give vitamin K and bile salts per os, or better still, 2 mgm. of the synthetic vitamin K both preoperatively and post-operatively. Vitamin K should be continued for three or more days after operation if there is any sign of liver damage. If there is marked cirrhosis of the liver, the blood prothrombin may fall despite the most intensive treatment with vitamin K. Release of obstruction does not immediately correct defects, and Ravdin reports a case in which bile salts did not appear in the bile for seven days following operation.

Vitamin K has been found to be of value in haemorrhagic disease of the newborn. The prothrombin of normal infants is high at birth, but it falls rapidly for the first 24 hours, falls more slowly until 72 hours of age, then it begins to rise gradually once more. During the first three days of life the plasma prothrombin may fall to below 5 per cent of the normal. When this occurs there is a definite bleeding tendency, and the infant is in danger of a massive fatal haemorrhage. The usual fall is to about 40 per cent of the normal level. A more marked fall indicates inadequate storage of vitamin K in the foetus. The strain of birth uses up these stores. Once the baby begins suckling the prothrombin rises since a bacterial flora is introduced into the intestine which can elaborate vitamin K.

Haemorrhagic disease of the newborn was twice as frequent in the clean, ideal surroundings of the New York Hospital as it was in the Berwind Free Clinic. The infants in the tenement houses had

a much better chance to infect their intestinal tracts with a beneficial intestinal flora, than the more privileged babies in the clean maternity hospital. Haemorrhagic disease of the newborn is self-limited but it may be fatal in the first few days of life. It is promptly cured by administering the synthetic compounds of vitamin K by mouth or intramuscularly. It can be prevented by giving the mother vitamin K for a few days before the child is born. Waddell advises giving it to the mother where long or precipitous labours are anticipated, and to the infant following difficult labour, especially if instruments have been used. This decreases the possibility of intracranial haemorrhage.

Conclusion

The chance observations of one man, Dam of Copenhagen, have brought to us, not only the explanation of previously obscure haemorrhagic diseases, but also the specific remedy with which to treat and cure them. Vitamin K and the synthetic compounds resembling it are of great value in the treatment of the bleeding tendency in obstructive jaundice, and of haemorrhagic disease of the newborn. The mortality from these two causes will undoubtedly be markedly reduced. We must remember that vitamin K has one function in the body, that of increasing the prothrombin level in the blood by its action on the liver. It is therefore of no value in the bleeding of haemophilia or thrombocytopenic purpura.

The Horse and Buggy Doctor

"Most of them followed their calling in the villages or towns that lie among the hills or along the inland streams. Only those who have lived the kindly, mutually dependent life of the country, can tell how near the physician who is the main reliance in sickness of all the families through-out a thinly settled region comes to the hearts of the people among whom he labors, how they value him while living, how they cherish his memory when dead. For these friends of ours who have gone before, there is now no more toil; they start from their slumbers no more at the cry of pain; they sally forth no more into the storms; they ride no longer over the lonely roads that knew them so well; their wheels are rusting on their axles or rolling with other burdens; their watchful eyes are closed to all the sorrows they lived to soothe. Not one of these was famous in the great world; some were almost unknown beyond their own immediate circle. But they have left behind them that loving remembrance which is better than fame, and if their epitaphs are chiselled briefly in stone, they are written at full length on living tablets in a thousand homes to which they carried their everwelcome aid and sympathy."

"Currents and Counter-Currents in
Medical Science."
Oliver Wendell Holmes,
Houghton, Mifflin and Co.
1860

THE SYNDROME OF HEPATITIS AND SPLENOMEGALY

By I. P. WEINGARTEN '41

Introduction

There is a group of pathological conditions characterized by hepatitis in association with splenomegaly. Falling into this group are the following conditions:—

(1) *The ordinary type of portal cirrhosis.* Portal cirrhosis may be found as feature of some other conditions e.g. hemochromatosis, Wilson's disease, Grave's disease; but such will not be considered in the paper.

(2) *Biliary cirrhosis:* this may be divided into two types:

(A) *Hypertrophic biliary cirrhosis of Hanot.*

(B) *Obstructive biliary cirrhosis.* This latter condition is the result of chronic obstruction of the bile ducts. Any change that results in the spleen is not uniform or constant. For this reason, this condition will not be considered further.

(3) *Banti's syndrome:* this term is used to include:

(A) *Splenic anemia.* This may be defined as a chronic disease characterised by splenomegaly, anemia and leucopenia, and a tendency to hematemesis.

(B)*Banti's disease.* This term is reserved for that group of cases which show at first the signs and symptoms attributed to splenic anemia, namely, splenomegaly, anemia and leucopenia, tendency to hematemsis, but which at a later date develop portal cirrhosis.

In the conditions that will be discussed (*portal cirrhosis, hypertropic biliary cirrhosis and Banti's syndrome*) the etiology is obscure. It has been suggested that we might conceive of a group of conditions in which as result of some toxic agent, a parenchymatous degeneration accompanied by fibrosis occurs. It may be first in the spleen, or first in the liver, in the former case giving rise to Banti's splenomegaly and in the latter giving cirrhosis of the liver.

Clinical Features

A detailed presentation of the various signs and symptoms of the conditions discussed may be found in any recognized text-book of medicine and need not be repeated here. A few essential features of value in differential diagnosis will however be given.

(A) *Portal Cirrhosis;* usually appears after age of forty; jaundice absent or fleeting; symptoms of portal obstruction; congestion of gastro-intestinal tract producing nausea, anorexia and even vomiting; hematemesis; melena, ascites; secondary anemia; by far the commonest condition.

(B) *Hypertrophic Biliary Cirrhosis;* occurs in young individuals; persistent jaundice; absent of signs of portal obstruction; leucocytosis; febrile attacks of abdominal pain.

(C) *Banti's Syndrome;* young subject; splenic enlargement appears first; when liver is cirrhotic a careful history of the order of appearance of the symptoms must chiefly be relied upon to demonstrate the primary trouble; secondary anemia and neutrophilic leucopenia; hematemesis.

Clinical-Pathological Discussion

(1) *Portal Cirrhosis*

The size of the liver in ordinary cirrhosis varies greatly. It may be smaller than normal and is then spoken of as atrophic, or it may be very large. There are all grades of transition between a small and a large multilobular cirrhotic liver. The large size of some cirrhotic livers may be due to considerable fatty change in the liver cell. The weight of the liver in portal cirrhosis may be normal, diminished or increased, but it is less often below than above or of normal weight. In a series of cases reported by Price the liver weighed less than normal in twenty-seven per cent of his cases.

Clinically, the liver is more often enlarged than diminished in size. Foxwell considered that "in the large

majority of cases of hepatic cirrhosis at all stages of the disease the liver is felt below the ribs". It is far from universally true that large cirrhotic livers are fatty. In a decided minority of cases, chiefly in patients whose history shows no alcoholic excess, the liver is considerably smaller than normal.

In the gross the peritoneal surface of the liver is opaque. This opacity is not uniform and is largely due to subcapsular fibrosis. There may be vascular adhesions between the liver and adjacent parts especially the diaphragm. The surface of the liver is irregular (hobnailed) in appearance. On section the liver is tough and is divided into areas of irregular size by grey, slightly gelatinous looking fibrous tissue. The liver is much paler than in health and has a yellowish-brown colour.

Microscopically the so called multilobular arrangement is present i.e. areas of varying size composed of liver cells and separated by broad bands of fibrous tissue containing many bile ducts. These areas may be smaller than a liver lobule, but are usually several times larger, so that several lobules appear to be grouped together. There is no true lobular arrangement for the normal relation to the central vein is lost. The nodules are composed largely of new cells which show irregularity of size and arrangement. The connective tissue forms broad bands between the islands of liver cells and may be young and cellular or old and fibrous. Chronic inflammatory cells are present, sometimes in great numbers, but the chief feature is the increased number of bile ducts.

The spleen is enlarged in portal cirrhosis but though not so markedly as in hypertrophic biliary cirrhosis. According to Klopstock it is enlarged in eighty per cent of cases. The splenomegaly is due to two factors, namely, portal congestion and cellular proliferation.

Portal obstruction is a well recognized accompaniment of hepatic disease. Jäger has been able to demonstrate that chronic portal congestion may lead to an enlargement of the spleen, but to a degree not exceeding that of the initial congestion. It is found that the distended spleen may reach three times its normal weight. Jäger's work would indicate that chronic congestion cannot produce an enlargement greater than this. On this assumption an average adult spleen weighing 150 grams might enlarge to 450 grams by chronic venous congestion alone. Thus, portal congestion does not necessarily produce splenomegaly, but in certain circumstances it may bring about some enlargement, the maximum in the human subject being about three times the normal size. Splenic enlargement to more than 500 grams in an adult involves some factor other than mere congestion.

The splenomegaly in cirrhosis probably depends primarily on poisons setting up hyperplasia and inflammatory changes in the organ, so that it is readily overdistended with blood. Although chronic venous engorgement alone is not enough to cause enlargement of the spleen, it favours stagnation of poisons and further it supplies blood to distend the softened splenic blood sinuses. Hence, it may be concluded that splenomegaly is primarily toxic and is aided by increased venous pressure in the portal system.

The capsule of the spleen is often thickened and shows perisplenitis. The spleen is often adherent to the peritoneum covering the abdominal wall, diaphram and stomach. On section the organ is sometimes firmer than normal and fibrosed; in other cases probably as a result of terminal infection, it is soft and diffluent and occasionally areas of a extravasation of blood are seen.

Microscopically there is dilation of the venous sinuses which spreads to the vessels of the Malpighian bodies and is followed by secondary atrophy of the lymphoid tissue in the Malpighian bodies and periarterial fibrosis. Fibrosis of the spleen due to increase in the trabeculae occurs in cases of long standing. Siderotic nodules are present.

A consideration of the relation between the size of the spleen and liver by Rolleston shows that the weight of the spleen has no constant relation to that of the liver and that it depends on

other factors which may or may not be associated with increased weight and size of liver. In some cases especially those running a rapid course, both liver and spleen are enlarged. On the other hand in latent cirrhosis the liver is considerably enlarged whilst the spleen is either of normal size or but slightly heavier than normal. Again in fatal cases of cirrhosis, the liver is sometimes smaller than normal while the spleen is almost constantly larger and heavier than normal. Splenomegaly is an important feature of portal cirrhosis. It is almost constantly present in fatal cases of progressive cirrhosis. The amount of splenic enlargement has been regarded as an index of the severity of the cirrhosis.

(2) *Hypertrophic Biliary Cirrhosis of Hanot*

This condition is very rare, thus contrasting with portal cirrhosis. There is no reason to regard alcoholism as related to the disease in the way as it is to the common type of cirrhosis.

The liver is uniformly enlarged but one lobe may be more affected than the other. Perihepatic adhesions uniting liver to diaphram are not uncommon, but otherwise the surface is fairly smooth or at most finely granular. It is dark green in colour, and on section is firmer than normal. Microscopically the liver shows unilobular fibrosis. The connective tissue is delicate and infiltrated with inflammatory cells. There is no portal obstruction. In the late stages a secondary multilobular cirrhosis may supervene.

The spleen is much larger than in portal cirrhosis. Its weight is two to six times the normal weight whereas the liver seldom weighs greater than two or three times its normal weight. The spleen is therefore, relatively heavier than the liver. There are frequently perisplenic adhesions from local peritonitis and thickening of the capsule. In uncomplicated cases the spleen is firmer than normal. Microscopically there is fibrosis with distension of the sinuses with blood. The Malpigian bodies in an early stage are hyperemic; later they may undergo fibrotic atrophy. There is also some endothelial hyperplasia in the pulp.

(3) *Banti's Syndrome*

Splenomegaly is the first symptom to appear. The spleen usually extends down as far as the umbilicus and may fill the entire left side of the abdomen. Splenomegaly may be present for years without the development of any other symptom. In chronic splenic anaemia a terminal multilobular cirrhosis may supervene, probably as the result of poisons manufactured in the spleen. This is called Banti's disease. At first the liver is enlarged but later it becomes shrunken.

It will be recalled that in hypertrophic biliary cirrhosis in the late stages a secondary multilobular cirrhosis may appear. This cirrhosis is probably of splenic origin and is like that appearing in Banti's disease.

The spleen is greatly enlarged but retains its normal shape. It is of firm consistence. The capsule is thickened, sometimes greatly. An increase of fibrous tissue is also evident on the cut surface. The cut surface has a rather beefy appearance.

Microscopically a fine fibrosis is the most characteristic feature. The trabeculae are also thickened. The characteristic histological appearance of the spleen presents periarterial haemorrhage and fibrosis, siderotic nodules, perimalpighian haemorrhage or fibrosis and dilated venous sinuses usually with increase of collagen in their walls. The siderotic nodules constitute the most striking appearance in sections of the spleen. They are often visible to the naked eye as rusty yellowish spots on the cut surface.

Degenerative thickening and thrombosis of the splenic vein are frequent occurrences. These changes may also involve the portal vein. The splenic vein is usually greatly distended and there are often huge collateral channels connecting the spleen with the stomach and the diaphragm. The intimal hypertrophy of the veins is not of the histological type usually associated with atheroma. The findings are thickening of the intima by increase in the number

of elastic laminae interspersed with loosely arranged connective tissue. The findings correspond rather to those occurring in the arteries in high blood pressure.

It is suggested that high blood pressure in the portal venous system plays an important part in producing the clinical and pathological picture of Banti's Syndrome.

The changes in the liver are not distinguished in any way from the common portal cirrhosis already discussed. However, even before portal cirrhosis appears, narrowing of the branches of the portal vein in the liver can be demonstrated and probably gives rise to considerable back pressure in the portal system.

The Relation of Symptoms to Lesions

The pathological features having been considered in some detail, we may attempt to correlate the symptoms and the lesions.

In the syndrome of hepatitis and splenomegaly the involvement of the two organs is probably simultaneous and varies only in degree. We are still in the dark as to the causation of the hepatitis and it is possible that there are many and varied factors at work, but the ultimate anatomical effects seems to be the same.

Splenomegaly may occur in association with hepatitis no matter how produced. Thus splenic enlargement has been observed in experimental hepatitis (Cameron and Saram) and also clinically in cases of acute and subacute yellow atrophy of the liver, ascending cholangitis with hepatitis, catarrhal jaundice, Weil's disease. The splenomegaly is brought about by two factors, namely venous congestion and cellular proliferation within the spleen. It appears that the proliferative change is in some way related to the hepatitis. The most active type of hyperplasia in the spleen is seen in association with acute inflammation of the liver.

The chief effect of portal cirrhosis is portal obstruction. There is congestion of the entire portal circulation with digestive disturbances, anorexia, etc. The ascites develops owing to transudation through the walls of the mesenteric veins. Because of the great decrease in size of the vascular bed, a collateral circulation is established, with the systemic circulation. Varicose dilations are apt to develop at the points where the two circulations communicate. Thus one gets oesophageal varices which may give rise to hematemesis, and enlargements of the hemorrhoidal veins giving rise to blood in the stool. Jaundice may develop in the terminal stages and is probably due to extreme disorganization and distortion of the normal architecture of the liver.

In hypertrophic biliary cirrhosis the process is concerned with the bile ducts and therefore there is no portal obstruction. The jaundice is probably due to a combination of hepatitis and partial obstruction to the flow of bile. If secondary multilobular cirrhosis supervenes, portal obstruction may occur.

In Banti's syndrome many of the symptoms can be explained on the basis of portal hypertension. The splenomegaly is partially due to this increased portal pressure which causes dilation of the sinuses, hemorrhages, indirectly fibrosis and partially to cellular proliferation within the spleen. Gastric hemorrhages can also be explained by the portal hypertension, as can the degenerative changes in the portal and splenic veins. The anaemia appears to be related to the lesions in the spleen, although in portal cirrhosis progressive anaemia is the rule.

Summary

(1) The etiology of the syndrome of hepatitis and splenomegaly is obscure. A likely explanation is to be found in the suggestion that the lesions are due to some toxic agent acting on the liver and spleen. The involvement of the two organs is probably simultaneous and varies only in degree.

(2) In portal cirrhosis the liver and spleen are frequently enlarged and palpable, although only one or neither may be enlarged. In hypertrophic biliary cirrhosis the liver and spleen are almost always markedly enlarged. In

Banti's sydrome the spleen alone may be enlarged (splenic anaemia); or first the splenomegaly may appear to be followed at a later date by clinical involvement of the liver (Banti's Disease).

(3) The microscopic picture found in the spleen is similar in all three conditions. The microscopic picture found in the liver in portal cirrhosis is in no way distinguished from that seen in Banti's disease and is very similar to that found in late hypertrophic biliary cirrhosis when multilobular cirrhosis has supervened.

(4) Portal cirrhosis is a disease of middle life, while Hanot's cirrhosis and Banti's Syndrome are found in the younger individual.

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On the 3 Senses, Memorie, Imagination, Reason

"On the contrary the Arabians will have, that every one of these faculties enjoy his proper seate; and they have very goodly reason for that which they holde. First, it is very certaine, that there are diverse pettie chambers in the braine, which the Anatomists call Ventrecles, these chambers are not for nothing, yea and there is no man that can thinke, that they were made for any other use, then to be lodgings for these three faculties; and that, as though the imagination should be lodged in the two first, Reason in the middlemost, and Memorie in the hindermost; and the appearance of the truth of this thing is very great; for the imagination receivth all the objects of the senses; but so the case standeth, as that all the senses are in the forepart of the head: the imagination presenteth all these objects unto the reason, which maketh them voide of matter and universall, so that of necessitie it must follow as second. The reason having after some time served and satisfied it selfe of these pleasant formes, committeth them to the custodie of memorie: whereupon it followed of necessitie, that it should be placed behinde, and as it were in her inner chamber. Furthermore, imagination being effected by receiving in of formes, must have his seat in the softest part of the braine, because the prints of pictures are most easilie set in a soft bodie; Memorie which keepeth and retayneth the formes, craveth a more hard part, for else the picture would be defaced by and by after that it should be printed: Reason, as the greatest of renowne ought to be placed in such a part of the braine, as is most temperate: so then we must beleeve that imagination is before, and memorie behind."

A Discourse of the Preservation
of the Sight; of Melancholike Diseases;
of Rheumes, and of Old Age.

by M. Andreas Laurentius
translated by Richard Surphlet
1599

(Published for the Shakespeare Association)
by Humphrey Milford
Oxford University Press. 1938.

MEDICAL ECONOMIC SERIES

[Editor's Note: These Bulletins are put out by the Canadian Medical Association, and are of the utmost importance. Two appear in this issue, and more will appear in the JOURNAL next year.]

FOREWORD

The practice of medicine is now undergoing radical changes. These changes are particularly noticeable in the economic relationship of the doctor to his patient. More, and perhaps more drastic, changes may be anticipated during the next decade or two. Therefore, it is highly desirable that every young doctor going into practice should have a clear conception of the issues facing the medical profession today and of the basic principles which should underly their solution.

For these reasons the Committee on Medical Economics of the Canadian Medical Association invited Mr. Hugh H. Wolfenden, F.I.A., F.A.S., F.S.S., to prepare a series of bulletins on Medical Economics which is now being issued for the use and guidance of senior students, interns, residents and young practitioners who desire a concise yet reliable review of this subject. In addition to being a noted actuary and authority on social economics, Mr. Wolfenden is Advisor on Medical Economics to the Canadian Medical Association and it is hoped that these authoritive bulletins, scheduled to appear from time to time, will be utilized as a basis for group discussions by the interns of hospitals, assisted by those staff doctors who are familiar with the subject.

In all the work of the preparation and distribution of these bulletins the Committee on Medical Economics and Mr. Wolfenden have been greatly assisted by Dr. Harvey Agnew, Secretary of the Department of Hospital Service of the Canadian Medical Association.

Additional copies of this and the bulletins to follow may be obtained from the Canadian Medical Association, 184 College St., Toronto.

Wallace A. Wilson, M.D.,
Chairman, Committee on Medical Economics.

BULLETIN I—THE DOCTOR'S INTEREST IN MEDICAL ECONOMICS

There are four terms or concepts which constantly recur in present-day discussions and which should be clearly understood at the outset:

(1) Medical Economics;
(2) The Economic Task of the Medical Profession;
(3) Health Insurance; and
(4) State Medicine.

(1) WHAT IS "MEDICAL ECONOMICS"?

This subject has been well defined recently as "the study of the production, distribution, consumption, and cost of the various types and kinds of medical services that promote and preserve the health of the people, with due regard for the ethical concepts of the medical profession, the highly confidential nature of the profession's services, and the necessity for maintaining always flexible procedures which can take into account the spiritual elements and the personal characteristics of the individuals concerned".

This definition emphasizes the following important points:

(*a*) *The ethical concepts* which form the background of medical practice *must never be sacrificed to economic pressure* from any source;

(*b*) *The confidential nature of the relationship between patient and physician must be maintained*;

(*c*) *The pressures of economic problems*, whether those pressures are felt by doctors or by their patients, or by both, *must not result in the diagnosis and treatment of disease being approached as a mass "efficiency" problem* without regard for the individualistic elements involved;

(*d*) *Any "planning" of an economic type must be flexible*, so that it will not submerge spiritual and personal values.

(2) HAVE WE AN ECONOMIC TASK?

The medical profession has an "economic task". This may be defined as "the development and provision of the best possible health services—both preventive and curative—for *all* the people, *at a price which is fair to all the people, including those who render the services*".

Some of the phrases in this definition should be given supplementary interpretations:

(*a*) *The "best possible health services—both preventive and curative"*, does not necessarily mean the best attainable anywhere. As a practical matter, it evidently *must mean* only *the best services which can be obtained for an individual under the particular environments of that individual, his available physicians, and others who may be called upon to render services, and with due regard to the available and attainable facilities.*

(*b*) *"For all the people"* must take into account the distinct positions of the various social-economic groups, such as (i) the unemployed without means; (ii) the unemployed with small independent incomes (from investments, pensions, etc.); (iii) the employed with inadequate or low incomes; (iv) the employed and unemployed with modest incomes up to perhaps $1,800, $2,000, or $2,400 per annum; (v) the upper middle class, both employed and independent, with incomes above the $1,800, $2,000, or $2,400 level, but below about $4,000 or $5,000 per annum; and (vi) all those with incomes above the preceding $4,000 or $5,000 per annum. In particular it should be realized that *the problem of providing the best possible health services will not be dealt with adequately by adopting measures (e.g., health insurance) only for arbitrarily specified economic or employee groups* (such as those in groups (iii) and (iv), as is so often suggested).

(*c*) What is a "fair price"? This is not easy to state. *The fairness of a price may be* (though not necessarily) *determinable to some extent by the patient's ability to pay.* It is equally important, however, that the payments received by the doctor shall be fair to *him*, and that payments similarly received by all others who render services shall be fair to them. The ideal formula for medical payments is not likely to be discovered by any kind of arbitrary action—whether such action be taken by governments, patients, or the profession itself. All such economic figures must remain, in the last analysis, a matter of negotiation and agreement, founded on reasonableness, fairness, and good sense.

"Health Insurance" and "State Medicine" will be defined and elaborated in Bulletin No. 2 of this Series.

Students will find (if they desire it) additional information on these matters in pp. 1-3, 7-10, 11-17, *et al.* of the Canadian Medical Association's recent publication entitled "The Canadian Medical Association and the Problems of Medical Economics".

BULLETIN II—WHAT IS HEALTH INSURANCE?

(3) HEALTH INSURANCE

The fundamental concept of any kind of insurance is very simple in principle, although in practical application its administrative details may be complex. The fundamental concept of insurance is "the co-operative association of a large number of persons, who agree to share amongst themselves the burdens resulting from the occurrence of a particular contingency—such as the occurrence of death, sickness, unemployment, etc.—by the payment of the necessary contributions into a common fund, from which benefits, related strictly to those contributions, are distributed in alleviation of the burdens against which the insurance is effected".

It is important to note that:

(*i*) Insurance requires the co-operation of a *group* of persons—it is a device for sharing burdens, not for shifting them;

(*ii*) *The benefit* which may be claimable by any member of the insured group on the occurrence of the contingency against which the insurance is effected (i.e., the occurrence of sickness, in the

case of health insurance) *may be more or less than the total contributions of that particular member with interest and after deduction of expenses*;

(*iii*) *Insurance may be "participating"*, in which case the member may receive "dividends", equitably apportioned, if the actual financial experience of the plan is more favourable than that originally anticipated, or *"non-participating"*, in which case the member will receive his contracual benefits but nothing more.

What Is Necessary to Ensure Satisfactory Operation?

Insurance being thus a co-operative group effort in which all will share as their respective interests, equitably determined, may require, means that the following conditions must also be satisfied in order that the insurance plan may be workable in practice:

(*a*) *The contingency must be capable of definition, understanding, verification, and practical interpretation.*

In the case of health insurance this means, in the first place, that the types of sickness to be covered by the insurance plan must be clearly defined in the insurance contract. The types of sickness so covered, and those which are excluded, must then be set out in the contract in terms which the layman and the Courts can understand. The machinery of the insurance plan must also make adequate provisions to allow its administrators to verify (i.e., to investigate and check) every claim for benefit which may be made, in order to make sure that the claim is legitimate, and that it falls within the terms and conditions of the plan. Finally, all the definitions and procedures thus understood by the insured person and the administrators of the plan must be capable of reasonable and sensible interpretation, so that the plan may work successfully in practice without developing frictions and dissatisfactions.

(*b*) *The contingency must involve a loss which can be evaluated*;

(*c*) *The probability of the occurrence of the contingency must be predictable* within the limits of variation indicated by the laws of chance, and *the contingency must be one which is not likely to occur simultaneously to every member* of the insured group, *and which will not be influenced unduly by the voluntary acts of those insured* (such as, for example, the incendiarism in fire insurance, or malingering in health insurance);

(*d*) *The group must be reasonably homogeneous* (in the sense that the persons must be sufficiently alike in respect of condition of health at entry into the plan, family history, domicile, occupation, etc., that the necessary rates of contribution for each age and sex can be calculated without the intrusion of anomalies or inequitable relationships between the various persons insured);

(*e*) *The contingency, the contributions, and the benefits must be clearly defined and understood*, so that claims can be investigated, certified, and controlled to make sure that they fall within the terms and conditions of the plan.

Sickness Insurance Can Be Practicable

Experience over the last 140 years—since the beginnings of voluntary sickness insurance societies in Great Britain and Europe—has demonstrated conclusively that insurance against the contingency of sickness can be considered as sound and workable provided the preceding conditions are laid down at the inauguration of any insurance plan, and so long as they are maintained throughout its existence. Its successful administration, nevertheless, is full of pitfalls for the unwary, and should be undertaken only by experienced persons.

The statistical and financial calculations which have to be made from time to time (similar in principle to those which are equally necessary in connection with life insurance, for example) are undertaken by an "actuary"—"the professional man

who is charged with the duties of performing all the statistical, mathematical, and financial calculations which form the basis of any plans involving the contingencies of human life" (birth, death, sickness, accident, disability, unemployment, etc.).

Safeguards to Ensure Actuarial Soundness

These calculations by the actuary evidently involve the consideration of some rather intangible influences which in turn are dependent upon the human behaviour of groups of persons. Actuarial procedure must therefore maintain safeguards and controls which will not permit any individual to exercise "selection" to his own personal advantage and against the equitable interests of the group of which he is a member. Since, moreover, the actuary is generally called upon to certify the "actuarial soundness" of health insurance plans, and as that term is frequently misunderstood, it may be well to state here the following as **the necessary conditions before any plan can be certified as "actuarially sound":**

(*i*) *The benefits offered must be defined, and the conditions for their payment must be clear*;

(*ii*) *The corresponding contributions* or other financial arrangements, by which the costs of such prescribed benefits are to be met, *must be determined by proper actuarial calculation*;

(*iii*) *Any power to alter the basis, terms, or conditions of the scheme must be subject to an actuarial certification* that the costs of such alteration are within the financial capacity of the plan;

(*iv*) *Adequate machinery must exist for the certification, inspection, and control of claims* for benefits, in order to make certain that they fall within the terms of the scheme, *and for the impartial and judicial interpretation of the* numerous and difficult *administrative problems* which inevitably arise.

It should be noted especially that one of the fundamental principles of sound actuarial procedure is to estimate details in advance for a limited period only, and to check them periodically against the experience which has actually emerged.

(4) STATE MEDICINE

State Medicine involves a concept wholly different from that of "insurance", and must be carefully distinguished from the latter. "State Medicine" means "a system of medical administration by which the state provides medical services for the entire population, or a large part thereof, and under which all practitioners are employed, and paid by the State on a salary basis or otherwise".

The essence of the method is, therefore, *State compulsion and control.* It presupposes, as a primary requirement, that the doctor (and others who would be called upon to render services) would be employed by, and would be under the direction and control of, the State, which under such circumstances would evidently be in a position to dictate procedures and modes of payment, and to prescribe and enforce penalties.

Feature Section

ALAN VAUGHAN, Editor

PHOTOGRAPHY IN THE PRACTICE OF MEDICINE

By C. E. SYLVESTER '41

At the risk of appearing trite, and then in the face of it, failing to practise what he preaches, the author wishes to begin this article with the quotation from Confucius: *One picture is worth a thousand words.*

Nowhere is this statement more applicable, than in the practice of Medicine. Probably the first words a City Editor says when giving an important news assignment is: "Get a picture". The same words might well be repeated by every student, interne, or practitioner when he is confronted with an unusual lesion or functional deficiency. Adequate and exhaustive though medical nomenclature may be, it still leaves a great many situations and facts confronting the doctor, crying for more graphic and dramatic expression. The author well remembers a moving picture of a pulsating aortic aneurysm shown him in third year during a physiology lecture but, ah woe! remembers not a word of the lecture that accompanied it. This example is cited not because it is the only one but because modern medicine makes such strides that in this country at least one is not likely to see large syphilitic aneurysms any more. Certainly the author has never seen one but feels that he would have a good chance of recognizing one with the picture impressed in his mind.

Numerous other examples of the value of photographs, both moving and still, for descriptive and record purposes could be offered by any of the readers of this JOURNAL. Recall the inspiration the coloured movie of the delivery shown in fourth year gave us, when the taste of the preclinical years was still in our mouths, and the glamour of the hospital not yet gone.

Fortunately nearly every large modern hospital already recognizes the place of photography as an important cog in an efficient machine, and maintains either a full or part time photographic department. The private practitioner, however, may not have access to these facilities when he needs them and although the aim may seem belated, it is the purpose of this article to describe how the practitioner of even less than average means may prepare and use adequate and useful photographic records.

Since moving picture equipment is more expensive and slightly more complicated to handle, its use is somewhat restricted for the private practitioner beginning his career and consequently this article will deal with *still* photography. The value of moving pictures must not be underestimated however, since *still* photographs cannot depict deformities of movement or moving processes.

The ordinary practitioner will probably confine his activities to three main types of photographs.

(1) *Still clinical photography*
  (*a*) *black and white*
  (*b*) *coloured*
(2) *Photomicrography*
(3) *Lantern slides*

Still Clinical Photography

Equipment—Obviously the most important item in this category is the camera itself. Theoretically any camera that will take pictures would do but because of

the practical limitations of lighting, a camera with a moderately fast lens should be chosen, a happy compromise being one of an aperature of F 4.5 or 6.3. If one has the bank account to stand it a lens of F 3.5 is ideal. Faster lenses are not necessary with modern flood bulbs and require more critical focusing. Since detail is all important the lens should be highly corrected, as most modern anastigmats are.

The shutter with such a lens described is bound to be adequate so long as it has an adjustment for instantaneous and time exposures. Those of the Compur type with speeds of 1—1/250 of a second, besides "time" are ideal. In order to get an image of adequate size which does not need enlarging and still is not too bulky the camera should be of a size taking film of at least 2¼" x 3¼" popularly known as 120, or, if plate holders and single cut films sheets are used 6.5 x 9 cm. Almost without exception the part being photographed is so small that even when the camera is as close as the focusing of the lens will allow, the image is still too small to show necessary detail without enlarging. To obviate this, a portrait lens which can be slipped over the lens of the camera, can be used. This lens serves the purpose of shortening the focal length of the camera lens allowing it to be placed much closer to the subject, with the resulting larger image still in focus. Double extension bellows serve the same purpose but are found only on the more expensive plate cameras. A suitable camera ranges in price from $10 - $70.

Some method of focusing must be used because, due to usually adverse lighting, the lens must be used open to its full capacity. This, due to an inherent characteristic of lenses, makes the distance over which a fixed object is in focus relatively small, and consequently focusing must be accurate. This can be done very easily by the use of a string knotted at convenient intervals corresponding to the graduations on the focusing apparatus of the camera. More accurate but more expensive methods involve the use of ground glass backs for the camera or else the use of a range-finder.

The next requirement is a suitable support for the camera. The ideal is a light but rigid folding metal tripod. These are so portable and inexpensive that any other method is a waste of time. The patient can become thoroughly exasperated waiting for you to pile chairs, old books and boxes under your camera, his temper only exceeded by yours when the whole mess falls down. Attempts at hand held time exposures or even moderately fast shutter speeds at close distances are doomed to failure. A tripod is a "must".

A filter or two serves as a valuable adjunct to the above equipment. Without going into the theory of light filtration the chief use of filters in clinical photography is to increase the contrast of detail. Those the author uses are a light yellow for use with orthochromatic (a red insensitive film) or a light green for use with panchromatic (a film sensitive to all colours). The filters are pieces of coloured optical glass which slip over the camera lens.

The next most important item is decidedly a lens shade. One should always be used. This small piece of apparatus is used, slipped over the lens or filter, and prevents extraneous light rays from reflecting in the lens components which cause "flare" spots on the finished picture. A very suitable and practical shade can be made for 10c by cutting off the business end of a wide base black rubber nipple.

Last but not least some form of exposure meter should be used to determine exposure time. The human eye is very fallible when it comes to judging light values. An exposure meter will save its cost many times over in film used, aside from the fact that the opportunity for a retake may never occur again. An electric meter is expensive but ideal. Visual meters are cheap ($1.75) and serve the purpose very well once their idiosyncrasies are learned.

Lighting—Because of the extreme variability of daylight and due to the fact that one's patient is almost invariably behind a door at the dark end of the room,

the use of artificial lighting solves this important problem and standardizes exposure time.

The simplest, most versatile and portable lighting units consist of spun aluminium reflectors which are attached to spring clamps. Into these are inserted two no. 1 (or for more light no. 2) photoflood bulbs. These provide adequate light for most purposes and give good colour values. The spring clamps make it possible to attach these lights to chairs, intravenous stands, screens, or even a conveniently standing nurse.

Light for clinical photography should come from two directions at about an angle of 45 degrees. This prevents the flat effect afforded by light coming from head on and provides modelling and form to the specimen or subject being photographed.

Procedure—Having gathered together the above, we are now ready to make our picture.

The amount of psychological technique needed to approach a kidney or other isolated pathological specimen is small and need be dwelt on no further. The approach to a patient however is extremely important. To burst in on a patient laden with equipment, rattling and banging like a one-man band is tactless to the nth degree. Pack your small apparatus in a fibreboard case and leave the whole shebang outside the door while you go in and explain to the patient that he has an interesting lesion which you would like to preserve for the edification of people who might not be fortunate enough to see a similar case. If you can get her, take the smiling nurse standing nearby in with you. Her presence adds reassurance especially if the patient is a female. Then with the equipment still outside the door, expose the area to be photographed no more than is necessary. Drape the redundant area with clean white towels leaving the lesion with a small portion of the surrounding normal tissue exposed in rectangular or circular frame of white towels. Reflections from moist surfaces are troublesome and often can not be avoided. Excess moisture can be absorbed by light application of a piece of gauze (not cotton wool). A small ruler with plain black figures should be included in the view to give an idea of size. Then, and only then, quickly set up lights, tripod and camera. Centre the lesion in the view-finder, obtain the proper exposure and take several negatives, possibly from several angles. Then retire quickly, murmuring appreciative words, and possibly with promises of a print when they are finished. The latter is especially valuable where progress of a process is being photographed and where a return trip is necessary. It might be mentioned here that the same tactfulness must be used towards the surgeon when photographing an operation. Be quiet and try to keep out of the way.

Development of the acquired films and the prints is the next step and is done according to standardized technique which cannot be gone into here. If you have not the facilities to do developing and printing by all means let the drug store do it. The production of the prints however is just as fascinating as the making of the negatives and provides a much needed hobby. Certainly there will be plenty of time during that oft bemoaned "First five years". Any print paper will do but a glossy surface is infinitely preferable since it gives desirable contrast.

Colour photography follows the same general procedure but unless special colour film compensated for artificial light is used (this is available at the same price), a compensating filter must be used. Dufaycolor is relatively cheap compared to Kodachrome but does not achieve the brilliant colours, nor is it as satisfactory for the necessary projection as the latter. Kodachrome on the other hand cannot be processed by the taker and also necessitates the use of a 35 mm. camera or else expensive cut film in a plate back camera.

Photomicrography

To achieve the best results with this branch of photography rather elaborate equipment is necessary. Nevertheless, with a little care and patience, very satisfactory results can be obtained without any other apparatus than that outlined, plus a good microscope and a proper light source.

To take a photomicrograph the desired area of the slide is brought into very careful focus, and with a light source consisting of a 100 watt bulb in a wooden box with an opal glass front. The light green filter is placed in the filter receptacle of the microscope (or over the camera lens). A point light source is used by the author because it gives more even illumination, but is more expensive. When the image is in the best focus possible, the camera on its tripod or otherwise clamped, is brought with its lens placed as close as possible to the eye piece of the microscope. A piece of black cloth is wrapped around the junction point (or the aforementioned rubber nipple over the eyepiece) to keep out extraneous light.

If a focusing scale camera is used it should be focused with a piece of ground glass over the film area (ground surface next to lens) or if this is not practical the focusing scale can be set at the 100 ft. mark. Plate back cameras are the most satisfactory for this purpose since they have an incorporated ground glass. It is difficult to recommend any precise exposure time and this is best found empirically for any one set up, and the times recorded for future use. It is best to make an exposure at say from 1–5 seconds and then make one at 5 times exposure and one 5 times less. It should be remembered that as magnification increases so also does exposure time.

A medium speed orthochromatic film should be used such as Eastman Verichrome. Reds will thus show dark in the finished print. Development of the negatives and prints proceeds as usual except that a contrasty developer and paper gives a more brilliant result.

Lantern Slides—These are the "bread and butter" production of the photographer-physician, especially if he does much lecturing or teaching in which their value cannot be overestimated. Nothing relieves the monotony of a lecture or demonstration like a few clear slides, as we who are still going to school well know.

Their actual production is exactly the same as the making of any contact print, since that is what they are, with the only exception that the emulsion is on a transparent glass base. To make them permanent, undesired areas are masked out with black paper and a clean cover glass of the same size, bound down at the edges with binding cloth or the new cellulose scotch tape. They are projected upside down, emulsion side towards the screen.

Summary

In the foregoing words the author has tried as briefly and simply as possible to outline the equipment and procedure necessary for the student and practitioner to do his own clinical and scientific photography. No pretenses are made as to the exhaustiveness of the description since many pages could be written on details of equipment and technique. It is the author's only hope that illusions as to complicated procedures are brushed aside and that the incentive to use photography as a means of permanent record has been provided. Make a beginning and no further urging will be needed. I guarantee there will be no limit to the many fascinating highways and byways photography will provide, whether they be medical or otherwise.

THE CANADIAN ASSOCIATION OF MEDICAL STUDENTS AND INTERNS

Resolutions passed at the Fourth Annual National Convention held at McGill University, March 14-16, 1941.

1. STUDENT HEALTH.

Whereas, the establishment of an efficient health service is of prime importance to medical students, and

whereas, a survey has shown that the health services in operation at the various medical schools are not adequate in all details,

and whereas, it would be of definite value for statistical purposes to have standard requirements for a health service and record system,

therefore, be it RESOLVED,

(1) That the delegates attending this convention consider themselves responsible for bringing to the attention of their own authorities the deficiencies in their own health service as compared with the minimum requirements as set forth below.

(2) That the results of the recent survey of the health services be published.

(3) That the following be considered minimum requirements for an adequate health service for medical students:

(a) Physical examination of all students on admission.
(b) Routine urinalysis, Wasserman, and Schick tests on admission.
(c) Special diagnostic facilities when indicated.
(d) Immunization against Smallpox, Diphtheria and other diseases where deemed necessary, on admission or during the preclinical years.
(e) A tuberculosis control program consisting of:
    1. Chest plate and tuberculin test of all students on admission.
    2. Positive reactors to have chest plates taken annually.
    3. Negative reactors to be retested twice annually in the clinical years.
    4. Individuals whose reaction changes from negative to positive to have chest plates twice annually for a period of five years, or until graduation.
    5. Chest plates available for students with symptoms which suggest early tuberculosis.
(f) A dispensary or clinic service be available to medical students, and that records of services rendered be kept on the individual's chart.
(g) A hospitalization insurance scheme, preferable underwritten by the university so as to avoid the high cost of private insurance.

(4) That the CAMSI Committee at the University of Manitoba be instructed to prepare a standardized record form to be used by all health services for the purpose of obtaining statistics concerning the health of medical students across Canada.

2. LEGAL PROTECTION FOR INTERNS.

Whereas, duly qualified and licensed interns in many Canadian hospitals are not protected by membership in the Canadian Medical Protective Association, or by any other insurance against suits brought against them by patients treated in those hospitals,

and, whereas this would seem to be a responsibility of the hospital trustees,

therefore be it RESOLVED that the National Committee of CAMSI appoint a temporary committee to prepare a memorandum to be circularized among the hospitals concerned, requesting the hospital trustees to assume responsibility for the cost of membership in the C.M.P.A. of all their licensed interns; and, that insofar as non-licensed interns are concerned, they be protected by some other form of insurance.

3. AMENDMENTS TO THE CONSTITUTION OF THE CANADIAN INTERN BOARD.

(1) The Honorary-treasurer of the C.I.B. shall be authorized by the National Committee of CAMSI to make payments for expenditures which are incurred by the Secretary of the C.I.B. in its operation.

(2) The Secretary of the C.I.B. shall receive an annual honorarium of $50.00 for his services to the Board. This honorarium shall be paid to him by the Honorary Treasurer upon the completion of his annual reports and their adoption by the national committee of CAMSI.

(3) Internships affected. The operation of the C.I.B. shall be extended to include the first year post-graduate internships for graduates of medical schools who intern during their final undergraduate year. This is to take effect when and where the Executive of the C.I.B. finds it advisable to do so.

4. CLASSIFICATION OF INTERNSHIPS.

Whereas certain medical schools require one year of internship before granting a degree, and whereas this internship corresponds to the first year post-graduate internship of other schools, this has led to confusion in the nomenclature. The following nomenclature is suggested.

(1) Junior internship—the first year's residence in hospital whether as a graduate or a final year undergraduate in certain schools.

(2) Senior internship. The second year of residence in hospital whether this is the 2nd year after graduation, or immediately after graduation.

5. UNIFORM ACCEPTANCE DATE FOR SENIOR INTERNSHIPS.

To permit junior interns to obtain the internship of their choice we suggest a uniform application date and a uniform acceptance date for such internships.

6. RESOLVED that the C.I.B. be instructed to prepare a list of senior internships available in Canada and to provide each junior intern with a copy of such information before October 15 of each year.

7. RESOLVED that the C.I.B. be instructed to contact all hospitals in Canada who have previously not employed women interns to determine whether such policy will be altered in the near future, and to prepare a revised list to be sent to all undergraduate societies by the end of October of each year.

8. RESOLVED that the National Committee of CAMSI permit the universities of Montreal and Laval to organize a Quebec Intern Board along the lines of the C.I.B. to promote the scientific, medical and material interests of senior interns in Quebec.

9. MEDICAL COUNCIL OF CANADA.

Whereas all Canadian universities do not as yet participate in the plan whereby graduation and Medical Council examinations are combined, be it RESOLVED that the CAMSI recommend to the parties concerned that advantage be taken of this new provision of the Medical Council; and, if necessary to carry out this plan, that representation on the Council be extended to include universities not now included, and that other necessary steps which will effect the Dominion-wide application of this provision be taken.

10. VISUAL EDUCATION.

RESOLVED that a National Film Committee be set up, with a secretary in Montreal, and one member in each medical school, to facilitate the selection and distribution of good medical movies among the medical undergraduate societies participating.

11. STATEMENT RE MILITARY TRAINING.

Whereas the medical students of Canada are extremely desirous of doing their part in

Canada's war effort; and whereas it is the opinion of the C.A.M.S.I. that the basic military training which medical student have been required to take during the past year has interfered with the pursuit of the medical curriculum required by our universities. This tends to lower the standard of medical education and is therefore incompatible with our maximum war effort, and this conference believes that medical students while in school be provided with the opportunity to devote their energies to their medical studies, and that the curriculum be supplemented by courses on war medicine or special Medical O.T.C. in place of basic training.

12. COOPERATIVES.

Whereas Cooperatives for the purchase of medical text books are an important consideration in the financial background of medical students: be is therefore RESOLVED that the local C.A.M.S.I. committees investigate the possibility of setting up campus cooperatives along the lines of the one established at the University of Western Ontario, and, where this is impractical attempt to contact a company that would give a substantial discount on medical texts if given all the business of that school.

13. STUDENT LOANS AND BURSARIES.

Whereas the C.A.M.S.I. believes that every means should be sought of providing financial assistance to medical students,

and whereas student loans and bursaries have proven to be valuable means of so doing,

be it therefore RESOLVED that:

(1) The representatives of all the schools investigate the details of the student loan system at their school, and, depending on the need, attempt to put into effect plans such as those discussed at this National Conference, i.e., low interest bank loans guaranteed by the university, etc.

(2) The C.A.M.S.I. fully endorses the present schemes of undergraduate bursaries in the medical schools of Canada, and that every effort be made to encourage extensions of these programs both by local and national approaches.

14. PUBLICATION.

RESOLVED that the National Committee be responsible for the publication of the proceedings of this convention,

that this be done in a special journal if possible, or otherwise that the National Committee confer with the McGill Medical Journal as to the possibility of including the proceedings in an issue of that Journal.

In the event of a National Camsi Journal being established,
This Journal shall be the property of the C.A.M.S.I.
An Advertising manager shall be appointed by the National Executive.
The annual dues from each school will include subscriptions to the Journal.
The Editorial Board will consist of members of the C.A.M.S.I. committee of the school holding the National Executive.

15. RESOLVED that the next National Convention be held before November 15, 1941.

16. RESOLVED that the National Executive for 1941-42 go to Laval University.

17. RESOLVED that this National Conference of the C.A.M.S.I. believes that junior interns as defined above (graduates of McGill, Queen's, Toronto, and Western, or final year undergraduates of Laval, Montreal, Manitoba, and Alberta) should be provided with adequate facilities for study, and should be paid a minimum maintenance allowance of $25 monthly with two weeks paid vacation. This resolution shall be presented for ratification to all medical school undergraduate societies and all Canadian hospital intern committees, and the results of this survey will be sent to the hospital boards concerned.

18. RESOLVED that the National Executive be instructed to send a letter of sympathy to Lady Banting in view of the recent death of Sir Frederick Banting, formerly Honorary Chairman of this association.

Abstracts

R. L. MacMILLAN, Editor

Contributors { D. J. Breithaupt; J. G. Watt; E. A. Armour; M. T. Townsend; W. R. S. Wilson; Ronald D. Nash

Lead Poisoning

A Clinical and Experimental Study of the Factors Influencing the Seasonal Incidence in Children

By M. RAPAPORT AND M. I. RUBIN
Philadelphia

Am. Int. Dis. Child., 61: 251, Feb. '41

Of 30 patients admitted to the Children's Hospital, Philadelphia, with acute frank lead encephalopathy, 24 were admitted during May to September. One was due to lead fumes, and 4 of the remaining 5 gave a history of the inception of Vitamin D therapy a short time before the onset of symptoms.

Experiments were conducted on young rats, observing their weight increase and general condition, while they were fed a diet containing 1.5% basic lead carbonate.

The lead markedly reduced the rate of weight increase, which effect was most marked when Vitamin D or sunlight exposure were given during the period of lead ingestion. After the lead in the diet was stopped, Vitamin D or irradiation had practically no effect on the recovery. Increased environmental temperature did not affect the toxicity of the lead in the diet.

Patients with lead poisoning excrete abnormally large amounts of coproporphyrin III, and increased porphyrin excretion was found in the rats also, which continued more than 30 days after cessation of the lead containing diet. When porphyrin is exposed to light it becomes a powerful proteolytic poison. It was thought perhaps the exposure to light worked in this way, but as mentioned above, if the rats were exposed to light after the lead feeding was discontinued there was no very significant change from a control series.

The explanation offered for the seasonal incidence observed is that Vitamin D causes increased absorption of lead from the gut, with resultant severe symptoms, precipitating an acute attack of lead encephalopathy. In this respect lead is seen to act much like calcium, and it has been shown by Sobel that Vitamin D does increase the lead level in the blood.

D. J. B.

Intracranial Aneurysms

By J. C. RICHARDSON AND H. H. HYLAND

Medicine 20: 1, Feb. '41

This 80-page monograph by our former teachers, now engaged in war work, proves to be both illuminating and interesting.

Between January 1928 and December 1938, some 118 cases of spontaneous subarachnoid haemorrhage were seen at the T.G.H. along with 8 cases of large unruptured aneurysm.

Pathologically, of the 34 cases of massive spontaneous subarachnoid haemorrhage that were autopsied, 27, or 90% were due to a ruptured berry aneurysm, in 6 the bleeding point was not found, and 1 was an arterial angioma.

Other aneurysms—mycotic, arteriosclerotic, traumatic arterio-venous aneurysms, and syphilitic aneurysms were not seen in this series.

The location of the aneurysms in or near the circle of Willis is shown.

The contrast with primary intracerebral haemorrhage is best seen by the 88% mortality, with only 30% under 50 years of age, while subarachnoid haemorrhage has a mortality of 53%, with 60% less than 50 years old.

The cases are grouped as follows:

1. Spontaneous Subarachnoid Haemorrhage. Uncomplicated, from which there may be a complete recovery in 3 months.

2. Spontaneous Subarachnoid Haemorhage with accompanying Cranial nerve Paralysis.

3. Spontaneous Subarachnoid Haemorrhage with Intracerebral Haemorrhage, due to the aneurysm lying in close proximity to the brain tissue, most often the frontal or temporal lobe, and causing destruction of that tissue.

4. Large Aneurysms without Rupture, with symptoms due to their locality.

The onset of symptoms is usually sudden, with headache, stiff neck, vomiting, loss of consciousness, and occasionally convulsions. It does not seem to be related to undue exertion, and is thought to be a gradual tearing of the aneurysm wall.

Physical signs are discussed, as well as localizing signs which may often be present contrary to some published statements.

The differential diagnosis is considered as well as the immediate and ultimate prognosis. As mentioned before, ½ of the cases die, and there seems to be only a mild chance of recurring attacks.

Treatment is complete bed rest with good nursing care for at least 4 weeks after any headache, stiff neck or vertigo has gone, with a gradual return to activity with no strenuous effort for at least 4 months, to allow complete fibrosis of the blood clot.

In the acute stage, the head of the bed may be elevated a bit to reduce the intracerebral blood pressure, codeia and aspirin are given for pain, and bromide and chloral, or phenobarbital, for restlessness. The patient must be warned not to strain at stool, liquid paraffin being given several times a day, and a small enema every 2 days. Intravenous hypertonic saline may be useful to reduce acute oedema of the brain.

Cerebrospinal fluid drainage is done on admission for diagnosis, and subsequently only for acute exacerbations. Only a small amount is removed, slowly. Repeated punctures do not give any ultimate benefit, and are not without danger. If the patient is improving, spinal punctures will not speed up the recovery any.

In the cases of unruptured aneurysm, ligation of the aneurysm may be very difficult, but one case is cited of ligation of the internal carotid artery with success. The patient had had an occulomotor paralysis. The obvious danger of ligation is the possibility of producing hemiplegia.

D. J. B.

The Present Status of the Influenza Virus

By RONALD HARE
Connaught Laboratories, U. of T.
Canadian Public Health Journal
February 1941, 32: 49

The causative organisms of the clinical entity of "influenza" are discussed, from the ferret influenza caused by nasal washings of patients—the influenza "A" virus, the "B" virus, and other viruses such as that of African Rift Valley Fever, and lymphocytic choriomeningitis, all of which may cause "influenza". Similarly haemolytic streptococci, possibly pneumococci, the rickettsiae, and early stages of the exanthemata may produce a condition identical with influenza.

Against the early virus disease, immunity appears soon, with virus neutralizing and complement-fixing antibodies. The titre quickly dies away, so that immunity is perhaps not long-lived. Also there may be subclinical infection causing relative immunity.

The virus does not infect when injected

subcutaneously or intraperitoneally (only via the lung) and in the mouse causes a marked degree of solid immunity. However, the ferret does not develop the same degree of immunity, and the human is perhaps like the ferret in this respect. Influenza and distemper virus grown on developing egg media causes an increase in human antibody level far in excess of any other method, and may be a means of immunization against virus "A".

Passive immunity has not yet been tried on the human.

This is seen to be a timely article on a subject in which we may soon all be very interested.

D. J. B.

A Study of the Cellular Exudates in Bowel Discharges: Their Diagnostic Significance

By Z. BERCOVITZ

Annals of Internal Medicine, Feb. 1941

This useful article briefly reviews the information to be gained from the microscopic examination of the stools with particular regard to the cellular elements to be found therein. The chief diagnostic aid in this study is in the differential diagnosis between functional diarrhoeas and those due to organic lesions; the former never produce a cellular picture of any consequence in the stools, while the latter invariably do. The differential diagnosis of the organic varieties is also greatly aided, the distinction between amoebic and bacillary dysentery being a conspicuous example. The specimen is examined in a wet preparation, both unstained and after being emulsified on a slide in Loeffler's methylene blue. The examination should be under oil immersion. Three types of specimen are mentioned:

(i) The outside of a normal stool, preferably the last part to be passed. The cellular constituents are most numerous here, and those with an origin in the proximal parts of the colon are included.

(ii) The last part of the watery stool after a saline cathartic.

(iii) The terminal mucus passed after three successive normal saline enemata. The cellular elements to be found are epithelial cells, polymorphonuclear leucocytes, round or lymphoid cells, and endothelial macrophages. The appearance of these is described, and illustrated by photomicrographs.

The Problem of Blast Injuries

British Medical Journal, Jan. 18, 1941

A Case of Pulmonary Concussion Due to High Explosive

By HADFIELD AND CHRISTIE

Haemorrhage into the Lungs in Death Due to Trauma

By JOAN M. ROSS

Reports of Societies

By ZUCKERMAN, HADFIELD AND AL.

The problem of the pathology of injuries due to high explosive blasts, and the best form of treatment, is receiving considerable attention. There is no dearth of clinical material in Great Britain at present. These three articles consider the matter on the basis of clinical, pathological and experimental findings.

Apparently the effect of the explosion alone, even of a very large bomb, is not sufficient to prove fatal at a greater distance than thirty to forty feet from the explosion, and injuries seldom occur at more than fifty feet. The blast is, however, always accompanied by flying missiles which cause both external and internal injuries. The ear drums are usually damaged if the injuries are due primarily to the blast, and should always be examined.

The primary lesion of pure blast injuries is probably dilatation and congestion of the alveolar capillaries of the lungs. Haemorrhage occurs, whether due to diapedesis or trauma, and this is pro-

gressive in cases that survive for any length of time. The symptoms go on to those of lobar pneumonia.

The problem of treatment is not yet satisfactorily settled, and resolves itself into a decision whether to transfuse for the fall in blood pressure or to employ venesection for the pulmonary congestion.

W. E. A.

A Case of Multiple Arterial Thrombosis Occurring in a Professional Blood Donor

By L. C. MONTGOMERY
Montreal

C.M.A.J., March 1941

Of interest to all medical students who give blood transfusions so that they may take in the Meds At-Home, and such, is this case report of a donor who gave too many transfusions.

On March 4th he gave 375 c.c. and on March 9th he gave another 500 c.c. Immediately following this he went to a tavern and ordered a glass of beer. This is the last thing that he remembers. He became unconscious before he had time to take his drink. When examined in hospital he was deeply unconscious and showed a left hemiplegia. There was no evidence of peripheral arteriosclerosis or hypertension. He regained consciousness in three days, but was unable to speak. Fifteen days after his cerebral accident he had sudden, severe pain in the left upper quadrant of his abdomen. His spleen was palpable and tender. This pain recurred in four days. He had fever and leucocytosis. Nine days later he suddenly developed severe pain all over the abdomen, he called for a bedpan, but his bowels would not move. He obtained no relief from morphine. He was operated on for mesenteric thrombosis, but it was impossible to carry out any satisfactory surgical procedures and the patient died in twelve hours. Autopsy confirmed the diagnosis of multiple thrombosis.

There was no definite evidence of abnormality in the blood. His serological tests were normal, but this case raises the question as to whether frequent transfusions bring about changes in the haemopoietic system, and it would seem advisable to keep a close check on all donors, limiting the number of transfusions that they can give in a specified time.

J. G. W.

The Source and Prevention of Septic Infection of Wounds

By R. HARE, M.D., AND REBA E. WILLITS, M.D.
Toronto

C.M.A.J., March 1941

This article is an excellent account of original work done by Drs. Hare and Willits during the last year. They obtained material from all wounds admitted to the Toronto General Hospital, the Western Hospital, St. Michael's, the Hospital for Sick Children, the John Inglis Company and the Massey Harris Company. Swabs were taken before anything was done to the wounds, usually within a few minutes to two hours from the time of accident.

It was found that clothing is not contaminated with haemolytic streptococci. Projectiles are free from them also. In 244 cases, only one wound had haemolytic streptococci in it at the time of examination. Str. pyogenes enter the wounds two to ten days after injury. The percentage of infected wounds increases for each day after the time of injury. Therefore Str. pyogenes is of exogenous origin and its source is the nasopharynx of cases, convalescents and carriers, and pyogenic infections in human beings. Nurses, orderlies, internes, etc., who are carriers expel streptococci every time they speak, sneeze or cough, but not if they are breathing quietly with their mouth shut. The hands, clothing, bedclothes, pockets and so on of carriers are heavily infected with pathogenic streptococci. Such an individual can infect open

wounds if he handles them with unwashed, ungloved hands. Transfer may occur from purulent secretions of other infections by way of the hands of the dresser or surgeon, by improperly sterilized instruments, but also by contamination of the air from an open wound such as a burn, by vigorous shaking of the sheets in making the patient's bed, and banging of the pillow. Bed-pans, bathes, and urine bottles may transfer the infection.

The suggested measures for prevention of infection of wounds are:

(a) Prevent direct expulsion of Str. pyogenes from the nasopharynx by the wearing of a suitable mask covering mouth and nose. Also forbid all attendants who are carriers to go on duty until haemolytic streptococci are shown to be absent from their nose and throat.

(b) Clean sterilized gloves should be worn and renewed for every case. If this is impracticable, as in regimental aid posts or advanced dressing stations, unsterilized gloves should be donned, washed thoroughly with soap and water, then immersed in an antiseptic of adequate strength. If gloves are not available the hands should at least be washed thoroughly then treated with Dettol, phenol, etc.

(c) Isolate all patients who are nasopharyngeal carriers from the other patients.

(d) A patient with an infected wound should not be placed in the vicinity of patients with clean wounds.

(e) Clean, uninfected wounds should always be dressed first, before those which are infected. No part of the dressings of infected cases should be handled by the bare hands. The bedclothes should be disturbed as little as possible during the making of the bed. Floors should only be swept after a moist or oily dressing has been applied to keep the dust down.

J. G. W.

ATTENTION: Mr. W. R. S. Wilson recommends highly two articles appearing in the Amer. Journ. Surg., Vol. 51, March 1941.

(1) **Fifteen Years of the Tannic Acid Method of Burn Treatment** by Glover and Sydow:—Herein is discussed the various modifications of the coagulation treatment as they appear to affect the results.

(2) **Some Observations on Fractures of Long Bones in Children** by Beckman and Sullivan:—The frequency of the different fractures is given. The treatment and the results are discussed. The problem of epiphyseal fractures is stressed.

Physical Fitness in Terms of Physique, Development and Basal Metabolism

By N. C. WETZEL

J.A.M.A., 116: 1187, March 22, 1941

The author has devised a new method of measuring physical fitness in children by the use of a grid. This grid is a type of graph on which three variables are plotted, the weight, the height and the age.

Everyone is aware of the difficulty of estimating physical fitness by the use of the present height-weight tables. Normal variations are unknown and one is apt to dismiss the problem by saying well-developed, well-nourished. Auxiliary aids have been suggested such as measuring various diameters of the body, the depth of the skin and the vitamin levels in the blood. The grid system avoids all these complicated procedures and with a minimum of time and effort an accurate estimate can be made.

The underlying principle of this method is the fact that a person developing normally from infancy to maturity follows a narrow channel. This channel is constant for individuals of any one physical type. Channels have been drawn on the grid from actual observations so that all physical types are represented. They have been tested on a large number of children and found to be reliable.

A given child as he grows older should follow a certain channel but if he gets out of it a medical examination is indicated to find the cause. In this way a reduction

of physical fitness may readily be determined and early corrective measures instituted by the physician. About 30% of children are found to need medical attention.

An additional feature of the grid is that the basal metabolic requirements for a particular child may be read off and in this way an accurate diet may be constructed.

This article should be read by everyone. It is a very definite contribution to the field of preventive medicine and probably will be widely recognized in the future. R. L. M.

A Skin Test for the Diagnosis of Pregnancy

By F. H. FALLS, V. C. FREDA AND H. H. COHEN

Amer. Journ. Obs. & Gyn. 41: 431, 1941

These workers have developed a highly satisfactory form of skin test for the diagnosis of pregnancy. The material used consists of diluted colostrum from 28-week primiparous pregnant women.

The nipple is cleaned with ether and the material is expressed into a sterile test-tube. Equal parts of normal saline solution were added. To 10 c.c. of this mixture 1/10 c.c. of 1 : 100 methiolate solution is added as a preservative.

1/50 c.c. of the material is injected intracutaneously. If the patient is pregnant the wheal produced by the injection appears pearly, has no pink areola and disappears in one hour.

In non-pregnant patients the wheal soon increases in size and has a pink to red areola. The reaction grows in size for an hour and persists for 4-5 hours.

The authors observed:—

(1) An intradermal injection of colostrum solution gave no reaction in 98% of pregnant women.

(2) Non-pregnant women reacted to similar injections with the formation of the characteristic wheal and areola in 96% of cases.

(3) Males and children before the age of puberty reacted similarly to non-pregnant females.

It may be concluded, therefore, that the test is of value in the diagnosis of pregnancy. M. P. T.

Hemoptysis in Rheumatic Heart Disease

By WOLFF AND LEVINE

Amer. Heart Journ., 21: 163, Feb. 1941

A series of 521 cases with rheumatic heart disease are reviewed. Fifty or 9.5% of these patients had hemoptysis. The age of onset of the hemoptysis was 33.4 years. All but one out of the fifty patients had mitral stenosis.

It was observed that pulmonary infarction is the commonest cause of hemoptysis where congestive failure exists. In cases where no congestion is present the cause is mitral stenosis alone: the right ventricle forces more blood into the lungs than can get out through the stenosed valve.

Hemoptysis in patients with no cardiac enlargement has no prognostic significance but in those with cardiac enlargement it is very grave: two-thirds are dead within a year.

The authors point out that when hemoptysis occurs in rheumatic heart disease only rarely is it caused by something other than the heart disease. R. N.

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